CARDIAC ARREST

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IN spite of improvements in anaesthetic and surgical technique, the hazard of sudden and unexpected death during an operation still exists.

The circumstances of anaesthesia and surgery predispose to the development of depression and even cessation of the circulation and respiration. During the past decade there has been a widespread interest in "cardiac arrest during anaesthesia," as evidenced by the multitude of clinical and experimental reports published on this condition. In this literature there appears to be a considerable variation in the definition of "cardiac arrest." Palomera (1952) defines it as "the apparent cessation of the cardiac function manifested clinically by the lack of blood pressure and the disappearance of the apical and peripheral pulses."

The majority of people about to submit to surgery fear the anaesthetic rather than the operation. There exists in the minds of many a primitive fear of sleep from which they may never awaken and when sleep is to be induced by a general anaesthetic, that fear is increased, because no anaesthetic is without risk.

In a widening surgical field operations are now commonplace on the heart, lungs, oesophagus and even on the abdominal viscera by a path through the open thorax, with the added disadvantage of possible disturbances of cardio-respiratory function if the operations are not carefully planned. Further, operations are now carried out on the aged, many of whom have cardio-vascular and respiratory defects which render them subject to unusual risks. It is not surprising that deaths in the theatre from cardiac arrest occur and this in spite of great improvements in the technique of anaesthesia made possible by the advent of the specialist anaesthetist. The many benefits of modern anaesthesia are partly offset by accompanying disadvantages which increase the incidence of cardiac arrest during induction. In three out of the four cases here reported cardiac arrest occurred during the stage of induction.

Perhaps the rapid methods of induction in vogue today, whereby intravenous anaesthetics and muscle relaxants are used constantly and in combination, are responsible for the fatalities by sudden lowering of the blood pressure and by permitting early manipulations. This depression of the blood pressure and these premature manoeuvres may produce reflex disturbances of the heart leading to cardiac arrest at a time when physiological adjustments have not reached completion. These disturbances are in marked contrast to the gentler adjustments which marked the slower method of induction of yesterday. Cardiac arrest was then less common, an ether drip on an open mask being the anaesthetic most frequently used.

The functional activity of the cardiovascular system may be depressed or arrested during operation in several ways.

In the vast majority of instances death during operation is a sequel to anoxaemia resulting from respiratory insufficiency or simple asphyxia, the heart's action ceasing as a secondary effect.

Failure of the circulation may develop initially in the peripheral vascular bed or result from haemorrhage, cessation of the heart's action occurring as a terminal event.

In these two forms the onset of the impending cardiac arrest is invariably preceded by many warning signs of cardio-respiratory depression. The recognition of them will permit the institution of timely and life-saving measures before the heart's action ceases. Should cardiac arrest develop, resuscitative measures promptly and efficiently carried out may succeed in restoring cardiac function fully and permanently without damage to the central nervous system. Where sub-oxygenation has held sway for some time, preceding actual cardiac arrest, cardiac function may be successfully restored but the permanent cerebral damage which may have resulted from the preceding oxygen want may cause death within a few hours.

Of infrequent occurrence is the form which develops during what is regarded as a hitherto uneventful, uncomplicated anaesthesia. Although its incidence cannot be accurately stated, this sudden catastrophic derangement of cardiac function has been recognized in most large surgical clinics and is described as "primary" or "acute" cardiac arrest.

Electrocardiographic studies of the heart's action during anaesthesia and direct observation of the heart during intrathoracic procedures show that the arrest is characterized by the sudden onset of asystole or by the occurrence of ventricular fibrillation. The output of blood from the heart is suddenly stopped in both instances, in the former by cessation of cardiac action, in the latter by the change to ineffective, unco-ordinated and irregular contractions of the ventricular muscle. The effect of ventricular fibrillation is more serious than that of standstill since it is seldom, if ever, reversible.

Finally, in operations on the heart itself, the trauma of continuous manipulation may cause alterations of rhythm and rate which may terminate in cardiac arrest.

The division into primary and secondary types is of academic interest only and of no practical value. The two vital functions, respiration and circulation, are so dependent on each other that, if one fails, the other soon languishes. Neither can continue for any length of time without the other. From a practical point of view the condition would

be better termed "cardio-respiratory arrest" for successful treatment depends upon the understanding of this association and inter-dependence of circulation and respiration.

The responsibility rests largely with the anaesthetist. His burden may be added to by improper selection of cases or by the surgeon's attempt to do something for a patient, whose clinical condition indicates that his early demise is inevitable. In ordinary surgical procedures, there is little a surgeon does which may lead to cardiac arrest which is not preventable.

Experience shows that many deaths could have been avoided and it is becoming increasingly clear from accumulating knowledge that even if cardiac arrest occurs the position can often be retrieved.

Like any other living tissue, the heart is unable to function efficiently for long without oxygen and the brain cells are more susceptible still to oxygen want. Three to four minutes of complete anoxia is likely to produce irreparable brain damage. The cessation of an effective heart beat does not mean that it is irrevocable. Cardiac action may be restored after a long interval. Beck's dictum that "any normal heart can be made to contract" is within certain limits proved by experience.

Although cardiac massage has been carried out in Australia on isolated patients for many years with some success as may be gleaned from our literature, there have been many failures, almost certainly due to delay in commencing the massage. Until recently, when cardiac arrest occurred, resuscitation was usually left to the anaesthetist, whose persistent efforts met with occasional success. The surgeon was in most cases content to be a passive observer or merely lend a helping hand by performing artificial respiration or by injecting drugs in the hope of seeing the heart beat again.

AETIOLOGY

Many factors are thought to participate in the occurrence of central circulatory failure during operations under general anaesthesia, but the mechanisms and the interrelation of these factors are not always clear. Furthermore, as the cardiac and respiratory systems are so closely related, both anatomically and and physiologically, disturbance of one system very soon affects the other and the exciting cause becomes more difficult to find.

However, certain aetiological factors have been repeatedly demonstrated.

Circulatory failure may occur gradually, leading finally to cardiac asystole, or suddenly, leading either to cardiac asystole or (less commonly) ventricular fibrillation.

Factors which alone or in combination are strongly suspected are (1) oxygen lack (anoxia or hypoxia); (2) adverse effects of anaesthetic agents; (3) vago-vagal or vago-sympathetic reflexes; (4) haemorrhage; (5) malnutrition; (6) drug idiosyncrasy; and (7) toxaemia from accompanying infection.

(1) Varying degrees of oxygen lack (anoxia or hypoxia) can occur from respiratory depression by any anaesthetic agents. It may also occur from inhalation of vomitus or blood or from excessive secretion. It is seen of course in patients cyanosed before operation (congenital heart disease, anoxic pulmonary heart disease).

Oxygen lack may act in two ways. Firstly, since the metabolism of heart muscle is such that it cannot contract anaerobically, it may produce gradual cardiac arrest leading in sequence to bradycardia with associated hypertension, intraventricular block and finally cardiac arrest. In the second place, it may assist in causing the sudden onset of cardiac arrest. A physiological explanation of this is that a normal heart subjected to intense vagal stimulation, though arrest may occur at first, will soon resume beating. This is the phenomenon of vagus escape for which three explanations are given by Samson Wright. The first reason given by him is that the associated rise of pressure in the great veins causes reflex acceleration of the heart. The second is that the lowered arterial pressure tends to accelerate the heart reflexly via the sino-aortic nerves. The third is the assumption by the ventricles of their independent rate as they are not affected by the vagus *(vide infra)*. It is suggested by Reid *et alii* that a diseased ventricle, or one subjected to anaesthesia, cannot resume this "idioventricular" rhythm, that anoxia enhances the effect of the anaesthetic in depressing initiation of the ventricular impulses and conduction in the ventricles, and that this is the part played by oxygen lack in the development of sudden cardiac arrest.

This kind of cardiac arrest frequently occurs during induction of anaesthesia before the skin has been incised and when hypoxia and hypotension or other factors, e.g. toxaemia, are not necessarily present.

(2) The adverse effects of anaesthetic agents are hypotension, respiratory depression or a direct effect on nerve transmission. Barbiturates are repeated offenders in this regard. They produce hypotension by their central depressive action on the vasomotor centre and also by a direct relaxing effect on the musculature of peripheral vessels. Most barbiturates have a parasympatholytic effect. However, Goodman and Gilman state that the thiobarbiturates, of which the frequently used sodium pentothal is one, do not have such an effect and may indeed enhance cardiac responsiveness to vagal stimulation. Scoline, which is frequently used in combination with pentothal, produces relaxation of voluntary muscle and theoretically may therefore produce hypotension. It is conceivable that venous return may also be impeded by the absence of muscle tone, though these effects have not been confirmed by us.

Some drugs act directly on the heart (chloroform, cyclopropane, ethyl chloride, trichlorethylene), others by sensitizing the heart to adrenalin (cyclopropane, chloroform and barbiturates).

Certain drugs are used deliberately to produce hypotension during operation, the object being to reduce the risk of haemorrhage.

(3) Vago-vagal and vago-sympathetic reflexes.

The regulation of the heart rate and to some extent of its rhythm is mediated chiefly through the vagus nerve whose efferent fibres end in the sino-atrial node, the atrial wall and atrio-ventricular node. It is doubtful whether effective efferent fibres pass to the ventricles.

Afferent vagal fibres pass from the heart, the lungs, bronchi, trachea, aorta pulmonary arteries, larynx and pharynx, as well as from many digestive organs, including the oesophagus.

Stimulation of these structures arising as a result of anaesthetic or surgical technique may give rise to reflexes which affect the heart. Reid *et alii* suggest that these reflexes pass in one of three ways:

1. From a vagal afferent nerve via the vagus centre, then by a vagal efferent.

2. From one branch of the vagus to another without passing centrally.

3. By a vago-sympathetic route.

The first two reflexes presumably inhibit the heart while the third stimulates it.

(4) Haemorrhage, unless massive, should be a rare cause of cardiac arrest as blood transfusion is frequently given prophylactically during major abdominal operations, and invariably when a thoracotomy is being performed.

(5) Malnutrition as a cause of cardiac arrest has been commented on by Reid and his associates. Dr. W. A. Bye of the Royal Prince Alfred Hospital has had personnel experience of malnutrition as a cause of cardiac arrest. The following personal communication has been received from him:

"Army Medical Officers practising in Japanese prison camps in Malaya and Thailand during the 1939-45 War, observed cardiac episodes unusual in civilian experience. Under conditions of prolonged dietetic privation, exposure to tropical infections and physical hardship, sudden deaths occurred in comparatively young men. These deaths were not caused by disease of the coronary arteries. Less commonly, attacks of cardiac arrest occurred in hospitals. Some attacks, relatively short in duration, resembled those seen in the Stokes-Adams syndrome and terminated spontaneously. Others lasted longer, but appeared to have been relieved by injection of a solution of adrenalin into the heart muscle. In several cases in a hospital at Changi, this treatment was carried out on a number of occasions in successive attacks occurring over a period of days. These patients ultimately made a complete recovery. If the circulation could be re-established, it was the practice to give vitamin B1 by injection, provided adequate supplies were available. Attempts were then made to augment the diet as far as circumstances permitted.

"Attacks of cardiac arrest of still longer duration were seen. Although the heart began to beat again, death ultimately occurred due to irreversible cerebral damage. No electrocardiographic studies were possible under the primitive conditions of practice and the mechanism of the cardiac derangement is a matter for conjecture.

There can be little reasonable doubt that malnutrition was the basic cause. The term malnutrition is used in the widest sense. Although the common diet was often grossly deficient in vitamin B1, it was also as a rule deficient in many other components of the vitamin B complex and deficient in protein, fat and caloric value. Most of the patients described were markedly underweight and not infrequently suffered from recurring attacks of malaria or from some form of diarrhoea. Their complete recovery in some cases after several close encounters with death will be of interest to surgeons considering the conditions of cardiac arrest occurring under other conditions."

(6) Drug idiosyncrasy has been thought to produce sudden death. The effect of cocaine in this connection may be due to intense central vagal stimulation producing cardiac arrest (Goodman and Gilman). Two cases have occurred in recent years at the Royal Prince Alfred Hospital where spraying the throat of conscious patients with decicain was succeeded by sudden death. The same cause may have operated in these cases as with cocaine or alternatively they may be other instances of a vago-vagal reflex.

(7) Toxaemia from various causes may affect the circulation generally, rendering the myocardium or conduction tissue more vulnerable to anoxia, anaesthetic intoxication and nervous reflexes.

To confirm the presence of these adverse reflexes and to observe if possible the effect of hypotension on cardiac rhythm, certain patients with normal cardiovascular and respiratory systems who were undergoing abdominal operations, had electrocardiographic tracings recorded during the induction of anaesthesia, simultaneously with direct recordings of systemic arterial pressure and right auricular pressure.

Under local anaesthesia a Cournand arterial needle was inserted into a brachial artery and a cardiac catheter into the right auricle via the basilic vein on the same side. This was accomplished after premedication of the patient and immediately prior to operation, with practically no discomfort to the patient. The procedure is without risk. Similar anaesthetic drugs and technique were used to those employed in three of the reported cases (2, 3 and 4).
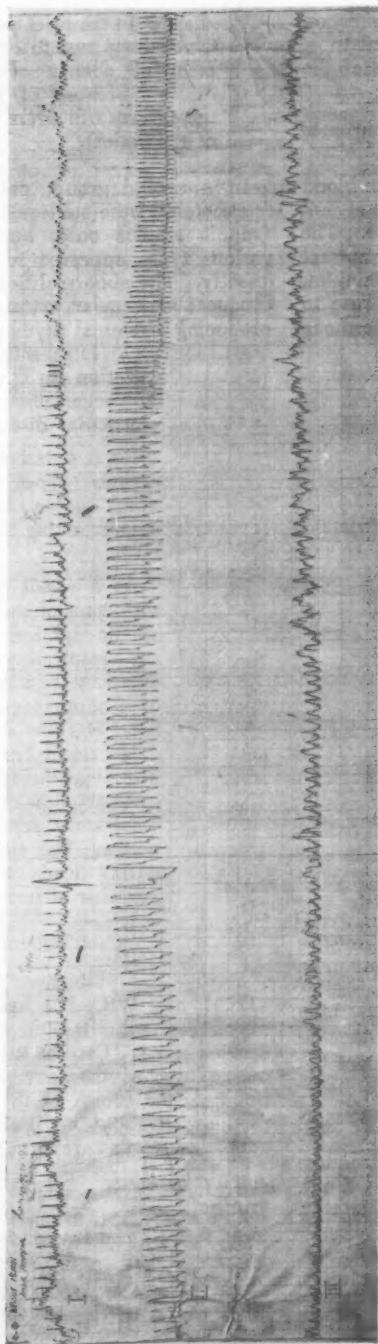
FIG. 1. Simultaneous electrocardiogram (above), directly recorded arterial blood pressure (centre) and right auricular pressure (below), recorded through a cardiac catheter.

The arterial blood pressure has fallen but has begun to rise about $1\frac{1}{2}$-2 min. after the injection of the pentothal (first mark on tracing).

The second mark indicates the time at which the laryngeal tube was inserted. A few seconds later a ventricular extrasystole has occurred.

The third mark indicates the time at which ether administration was begun. A second fall in blood pressure occurs soon after this.

There is a slight rise in right auricular pressure in the later half of the tracing. (See text.)

The effect of injected drugs, of insertion of the laryngoscope and endothracheal tube, of cyanosis and alteration of posture of the patients were noted (Figs. I-V).

This group of patients show the occurrence of the following events:

1. An invariable fall in systemic blood pressure with pentothal sodium, not necessarily increased by scoline. The blood pressure returned to normal within approximately two minutes. The blood pressure sometimes rose to a level higher than the pre-anaesthetic level.
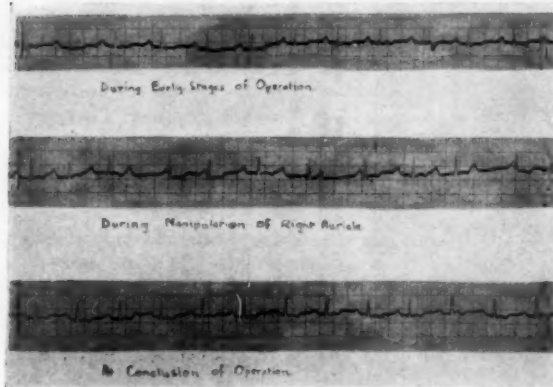

FIG. II. R.E., male aet 19. Three electrocardiographic tracings taken during the operation of pericardiectomy for constrictive pericarditis.

The second tracing shows atrio-ventricular dissociation during manipulation of the right auricle while pericardium was being dissected free. First and third tracings show sinus rhythm at stages before and after the atrio-ventricular dissociation.

2. A variable right auricular pressure accompanying the fall in systemic pressure. At certain times this remained at the pre-anaesthetic level, at others there was a slight rise. Proof was lacking that succinyl choline (scoline) was the cause of diminished venous return.

3. Occasional ventricular arrhythmias only (see illustrations). These arrhythmias were not noticed unless certain manipulations were carried out. These observations do not agree with the findings of Gruber et alii in certain anaesthetized animals. Cyanosis is difficult to assess because it cannot be produced experimentally in human beings. We did not observe adverse changes in the brief periods of cyanosis which happened to occur. The assessment of change of posture is also difficult because of interference with the electrocardiogram during movement of the patient.

In a second group, patients undergoing thoracotomy were subjected to electrocardiographic studies only, but arrhythmias of various types appeared to occur more frequently. The abnormalities noted were: (1) Frequent ventricular extrasystoles sometimes producing bigeminal rhythm; (2) first degree heart block; (3) intraventricular block; (4) variation in T wave voltage and ST segments; and (5) atrioventricular dissociation.

Representative examples of the various types of arrhythmias which occur are given in the accompanying illustrations (Figs. I-V).

As a result of our experience with cases of cardiac arrest we are of the opinion that hypotension and respiratory depression produced by the intravenous injection of pentothal enhance the likelihood of reflex disturbances which may result in cardiac arrest. The hypotension adversely effects coronary flow during diastole and respiratory depression causes hypoxia. The summation of these effects leads to defective oxygenation of heart muscle.

CASE REPORTS

During the past three years, two of us (J.S.M. and W.I.T.H.) have conjointly had experience in the treatment of four cases of cardiac arrest. Cardiac massage was carried out. Three of the four patients recovered, but one, the fourth of the series, has since died of advanced malignant disease. (See addendum to case reports.)

Case 1

R.P., a male, aged 57 years, was admitted to the Royal Prince Alfred Hospital for total laryngectomy.

Examination of his heart showed that he had mitral stenosis. His blood pressure readings were 130-80 mm. of mercury and blood haemoglobin content 15.5G per cent.

It was arranged that operation should be performed on 14th April, 1950. Anaesthesia was induced by trilene drip on an open mask and after intubation trilene and oxygen were administered by a semi-closed endotracheal technique. Whilst the skin preparation was being carried out, the patient's colour suddenly changed and his respirations and pulse ceased. Cardiac arrest was diagnosed and artificial respiration with 100 per cent. oxygen was commenced immediately. An intravenous injection of four cubic centimetres of Coramine was given without effect. An intracardiac injection of 0.3 cubic centimetre of a 1 in 1,000 solution of adrenalin and five cubic centimetres of a one per cent. novocaine solution produced no improvement.

After a delay of approximately four minutes, subdiaphragmatic cardiac massage was commenced and persisted in for ten minutes without any sign of spontaneous respiration or cardiac contraction.

Case 2

W.E., a male, aged 62 years, was admitted to the Royal Prince Alfred Hospital on 16th September, 1952, for operation for bronchiogenic carcinoma of the upper lobe of the right lung (Fig. VI).

No abnormality could be detected in his cardiovascular system. The electrocardiogram was normal and his blood pressure readings were 120-80 mm. of mercury.

The patient was taken to the theatre for operation on 22nd September, 1952. Anaesthesia was induced by the intravenous injection of one gramme of pentothal sodium and one cubic centimetre of succinyl choline. Oxygen was administered through the bronchoscope during intubation. The anaesthetic machine was now connected and gas and oxygen with minimal ether administered, following which

FIG. III.   The occurrence of frequent ventricular extrasystoles following laryngeal intubation. (See text.)
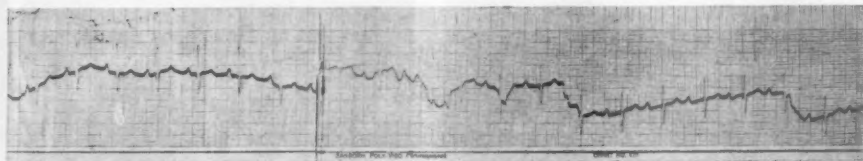

FIG. IV.   The tracings show a normal cardiogram in the early stages of the operation (left) with first degree heart block later (right).   (See text.)
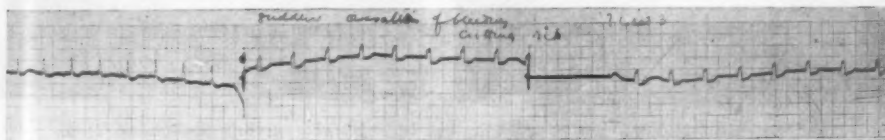

FIG. V.   The tracings show a QRS complex of normal duration (left), with prolongation of the QRS duration later in the operation.   (See text.)

COMMENT.

Acute cardiac arrest occurred due to the toxic action of trilene on the myocardium. Cardiac massage was carried out too late and subdiaphragmatic massage was ineffectual. The type of cardiac arrest, whether standstill or ventricular fibrillation, was not determined because no electrocardiogram was taken and direct inspection of the heart was not made.

a relaxant, ten milligrammes of d-Tubocurarine chloride, was given intravenously. Difficulty was experienced in adequately ventilating this patient, owing to spasm of the respiratory muscles.

After posturing the patient on his left side, his face became suddenly pale and his pulse imperceptible. Cardiac arrest was diagnosed and within one minute of its onset subdiaphragmatic cardiac massage was commenced. The exposure was made without any preliminary preparation and with ungloved hands.

One cubic centimetre of 1 in 1,000 solution of adrenalin was injected into the left ventricle through the chest wall and five cubic centimetres of one per cent. novocaine solution were injected intravenously. Five minutes from the onset of cardiac arrest normal rhythm was resumed.

On 20th October, 1952, the patient was again taken to the theatre and the operation of right upper lobectomy carried out. A drip transfusion of 0.4 per cent. novocaine solution into the veins was commenced before the induction of anaesthesia. The anaesthetic agents and technique used were exactly the same as those employed on the previous occasion when cardiac arrest occurred. The patient caused no further concern and was discharged from hospital on 20th November, 1952.

When seen on 16th March, 1953, he was clinically well but his eyesight was defective.

the lungs after intubation resulted in some anoxaemia. Manipulations around the carina and at the entrance of both bronchi and concentrated ether vapour blown in whilst the patient was lightly anaesthetized with pentothal sodium may have initiated vago-vagal reflexes which precipitated the onset of cardiac arrest.

Case 3

J.H., a female child aged 9 years, was admitted to the Royal Prince Alfred Hospital on 6th September, 1952, for surgical treatment for bronchiectasis (Fig. VII).

Examination revealed her to be a well-nourished child. Her heart sounds were normal and her blood pressure readings were 110-80 mm. of mercury. Examination of her blood revealed a haemoglobin content of 13.8G per cent. and her vital capacity was 1.3 litres.

Operation took place on 24th November, 1952. Anaesthesia was induced by the intravenous injection of 0.4 gramme of Thiopentone and 0.5 cubic centimetre of succinyl choline. After aspiration of bronchial secretions and intubation, a
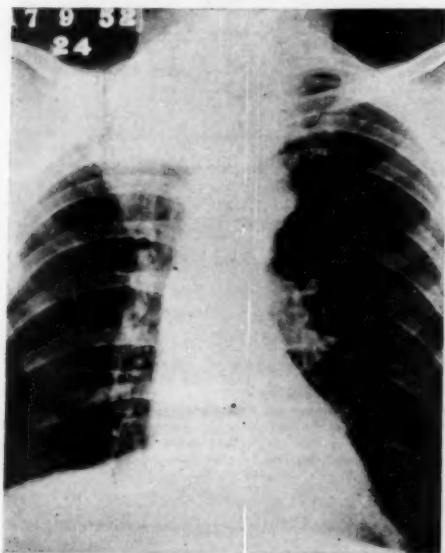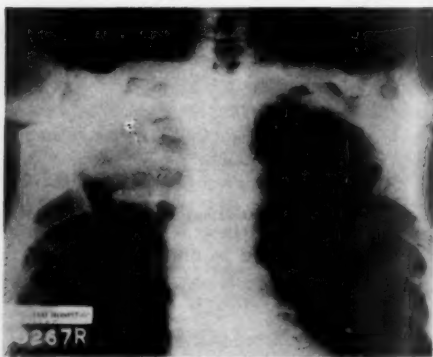

FIG. VI. Antero-posterior and lordotic skiagrams showing atelectasis with consolidation of the right upper lobe, consistent with the diagnosis of bronchiogenic carcinoma.

COMMENT

This patient developed acute cardiac arrest during induction of anaesthesia. We are of the opinion that the sudden change in posture of a patient manifesting hypotension caused by the induction of anaesthesia with pentothal sodium so lowered the blood pressure further as to interfere with the coronary flow and thus cause inadequate oxygenation of the heart muscle. The difficulty of ventilating

machine anaesthetic of nitrous oxide, oxygen and ether was administered with five milligrammes of d-Tubocurarine chloride.

The operation of right lower and middle lobectomies was then commenced. Folowing the ligation and division of one of the main vessels a ligature slipped and an acute blood loss of not more than 150 cubic centimetres resulted before the bleeding was controlled. The remaining arteries and veins of the lower and middle lobes were ligated and divided. Up to this stage the anaesthetic and operation had proceeded smoothly, but then it was noted that the

patient's blood was becoming dark. All operative manipulations were temporarily discontinued.

Aspiration of bronchial secretions and hyperventilation did not improve her colour. The heart was observed progressively to dilate and cardiac arrest occurred.

Extrapericardial massage was commenced and found to be ineffectual. An intravenous injection of one cubic centimetre of Coramine was administered and an intra-cardiac injection of one cubic centimetre of Coramine and 0.5 cubic centimetre of a 1 in 1,000 solution of adrenalin was given into the right auricular cavity. After a delay of 1½ minutes the pericardium was opened. At the first incision the heart was found to be grossly distended and in asystole, it bulged through the incision which was with difficulty enlarged. Intrapericardial massage was then commenced and seven minutes from onset weak, ventricular contractions occurred. Thirty seconds later, quite suddenly and dramatically, the

ophthalmoscope, but she was completely blind. She was allowed up on the tenth day after operation but her sight was still defective. When discharged from hospital on 18th December, 1952, her sight had improved to such an extent that she could recognize people.

Examined on 11th March, 1953, she answered questions intelligently. Her mother stated that she could detect no change in personality and regarded her mentality as being as good as before operation.

On 1st May, 1953, her eyes were examined by an ophthalmologist who reported: "Vision right eye less than 6/60; left eye, 6/12. No improvement with glasses. The pupil reactions were normal. The right visual field was markedly constricted and the left visual field showed some irregular peripheral loss. The optic discs, retinal vessels and fundi appeared completely normal. In my opinion the visual defect must be of cortical origin. Improvement improbable."
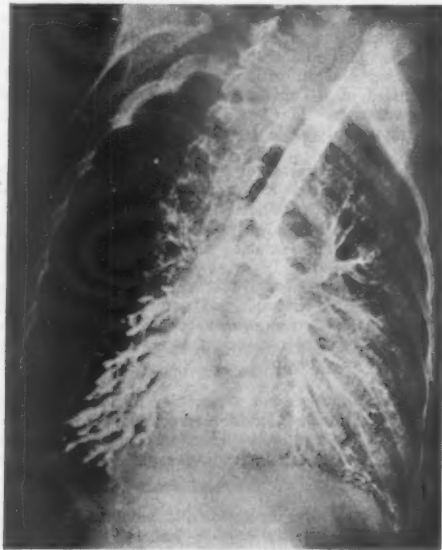

FIG. VII. Antero-posterior and oblique views of bronchogram showing bronchiectasis of the right lower and middle lobes.

heart resumed rapid, regular and complete contractions with improvement in the patient's colour. The operation was now proceeded with and completed without undue concern.

Artificial respiration had to be continued for one hour after the completion of the operation before automatic respirations became evident.

For two days she remained in a stuporous condition with muscle twitching and generalized rigidity. On the third post-operative day she recognized voices vaguely and would sometimes answer questions. Her fundi appeared normal on examination with an

COMMENT

This patient had cardiac arrest for a period of seven minutes from which she recovered with cerebral damage. She had a secondary cardiac arrest resulting from mild but prolonged suboxygenation of the blood and from cyanosis for a short time before its onset, which defied correction. Hypoxia was the cause of her cardiac arrest and was accentuated by a small acute blood loss at a time when oxygenation was subnormal.

Case 4

D.C., a male of 51 years, was admitted to the Royal Prince Alfred Hospital on 24th December, 1952, for thoracotomy. He had a bronchiogenic carcinoma of the right lung of doubtful operability.

He had heart sounds which were of normal quality but occasional extra systoles were audible. His blood pressure readings were 110-70 mm. of mercury. No pre-operative electrocardiogram was done. Examination of his blood revealed a haemoglobin content of 12.6G per cent. His vital capacity was 2.4 litres, 64 per cent. of normal, and his maximum breathing capacity 67.4 litres, 71 per cent. of normal.

He was taken to the theatre for operation on 12th January, 1953. Anaesthesia was induced by the intravenous injection of seven cubic centimetres of five per cent. sodium pentothal and 100 milligrammes of succinyl choline. During intubation a further seven cubic centimetres of sodium pentothal and 100 milligrammes of succinyl choline were given. The anaesthetic machine was now connected and ether and oxygen administered. Spasm and "bucking" occurred and in order to control these, 15 milligrammes of d-Tubocurarine chloride were injected intravenously. The heart beats immediately became irregular and feeble and cardiac arrest occurred.

The thorax was at once opened in the right fifth intercostal space. It was seen that the patient had an inoperable carcinoma of the lung. The pericardium was immediately widely incised and a small quantity of fluid, about one ounce, escaped. The heart was seen to be grossly dilated and in a state of ventricular fibrillation. Cardiac massage was commenced in less than two minutes from the onset at a rate of 60 compressions per minute. This was followed by the intravenous injection of 120 milligrammes of procaine.

Three minutes later ventricular fibrillation ceased and occasional, regular ventricular contractions followed. Six minutes from the onset of cardiac arrest the heart was beating rapidly and forcibly, but with extra systoles.

Two days after operation his mentality appeared normal and cardiac rhythm was regular. On the fourth post-operative day he was allowed out of bed and walked about the ward with no apparent ill effect.

He has subsequently died of advanced malignant disease.

COMMENT

This patient, suffering from advanced malignant disease of the lung and from mitral stenosis, developed acute cardiac arrest during induction of anaesthesia. We suggest that the following factors operated: (1) hypotension and respiratory depression; (2) anoxia from prolonged spasm and "bucking"; and (3) vago-vagal reflexes.

DISCUSSION OF CASES

In three of the four cases reported the patients developed cardiac arrest during the stage of induction of anaesthesia. Even in this small group of cases the frequency of cardiac arrest during induction conforms in general with that reported by other writers.

Three of the four patients were regarded as reasonably good "operative risks" in view of the nature of their disease. They had good cardiac muscle and perhaps for this very reason the type of heart arrest manifested by them was cardiac asystole. The fourth, a debilitated man with advanced malignant disease of the lung, mitral stenosis and coronary atheroma, developed ventricular fibrillation during the stage of induction.
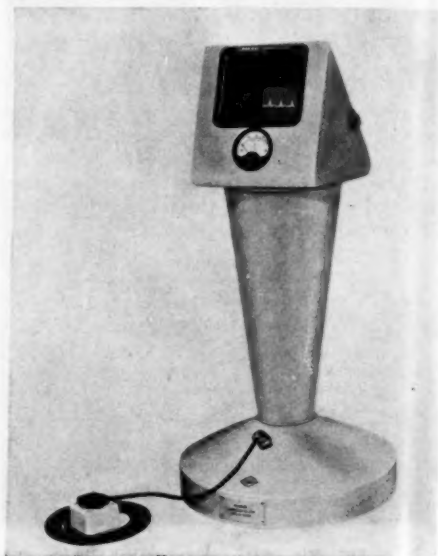

FIG. VIII. The cardioscope. A cathode ray oscilloscope which traces the electrocardiogram.

The four cases form an interesting group for study. Case 1 exhibited a true acute cardiac arrest, probably due to the toxic action of trilene on the myocardium. Vagovagal reflexes could have contributed. In Case 2 a sudden change of posture was the exciting factor in the production of acute cardiac arrest, following induction by the pentothal and scoline technique with its associated phenomena of hypotension and

respiratory depression. The patient recovered without damage to his heart muscle but with defective vision. Two weeks later he underwent pulmonary resection causing no concern during the operation. He exemplifies the fact that a patient with good cardiac muscle who has had cardiac arrest during induction of anaesthesia but recovers, may be successfully operated on at a later date. Case 3 could in general be regarded as one of secondary cardiac arrest, due to suboxygenation of the blood, terminating in acute hypoxia of the medullary centres and cardiac muscle. A small acute blood loss added to this by reduction of the volume of the circulating blood. After the patient's immediate recovery from cardiac arrest, the operation was continued as it had reached a stage when it could not be abandoned. That during the remainder of the operation she caused no undue anxiety illustrates the fact that operation can be proceeded with safely under these circumstances, provided that adequate oxygenation can be maintained. This child ultimately recovered but with cerebral damage affecting mainly her sight. In Case 4, the patient who was in poor physical condition, suffering from advanced malignant disease of the lung, which of itself may be accompanied by cardiac irregularities, and who had mitral stenosis and coronary atheroma, developed ventricular fibrillation during induction of anaesthesia by the pentothal-scoline technique. The same factors which operated in Case 2 may have played a part in causing the cardiac arrest in this case. It is of interest to record that such a debilitated patient recovered from ventricular fibrillation after treatment with cardiac massage and procaine injections.

A great deal of interest was attached to Case 3, in which the surgeon had direct observation of the heart during an operation through the open thorax, permitting the sequence of events leading to the appearance of cardiac arrest and the behaviour of the heart thereafter to be observed up to the point of recovery. These phenomena were characteristic and conformed in every detail to the description of other observers. In this patient the heart slowed and dilated, the beats becoming progressively more feeble until the dilatation was limited by the fibrous pericardium. The heart muscle gradually

weakened from oxygen lack until, unable to contract any longer against the rising intra-cardiac pressure, complete standstill in diastole occurred. The degree of dilatation was such that when the pericardium was incised, the heart literally bulged through it.

From observations made it appears that whilst the anaesthetist is actively engaged in bringing oxygen to alveolar cells, treatment should be directed by the surgeon towards emptying the heart and great veins and so reducing intra-ventricular pressure. This is best carried out by manual pumping of blood from the heart cavities and great veins to the arterial side of the circulation until oxygen is brought to the heart muscle and rhythmic contractions occur. Phlebotomy would reduce the pressure in the great veins and right side of the heart, but is not recommended as it is less effective and would have the added disadvantage of blood loss.

From the appearance of the grossly distended and cyanotic heart, it was difficult to see what effect adrenalin or other drugs could have in initiating contractions. The very appearance of the heart would substantiate the belief that the use of such drugs should be withheld until after cardiac massage had been commenced and cardiac distension and cyanosis reduced, when they would be of more use as a stimulant in initiating or augmenting feeble contractions.

Observations made during the treatment of these four patients drive home the view that cardiac massage is most effective when carried out by the intrapericardial route. The subject of Case 1 was the first patient on whom we conjointly carried out resuscitative measures. The delay was too long and the subdiaphragmatic route was ineffectual. The patient succumbed. In Case 2 the subdiaphragmatic route was again used but cardiac massage was promptly carried out. The result was successful as the patient had good cardiac muscle and the arrest occurred during induction. Where the cardiac arrest, as in Case 3, was due to suboxygenation of the blood, even though the patient was a child with good heart muscle, extrapericardial massage was of no benefit because it cannot be carried out with ease from the right side, but intrapericardial massage was effective. Where ventricular fibrillation exists, as in

Case 4, intrapericardial cardiac massage is an essential part of treatment, used in combination with procaine injections or a defibrillator.
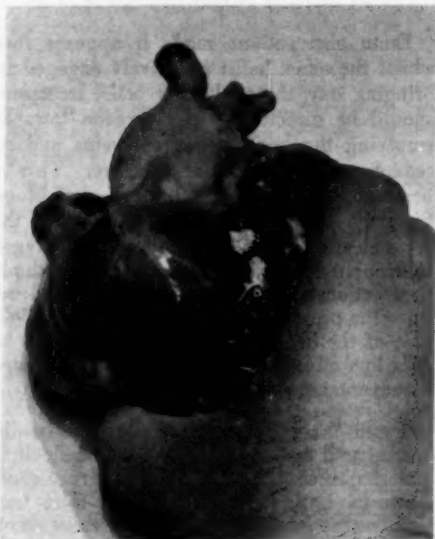

FIG. IX. The recommended method of handling the heart, showing a firm grip of the ventricles, leaving the auricles free.

PROPHYLAXIS

Cardiac arrest is an acute surgical emergency requiring immediate and exacting treatment if catastrophe is to be avoided. The possibility of its occurrence and the influences leading to it should be always kept in mind, as in many cases it can be prevented. From what has been said of the possible factors suspected of being related to its production, certain measures would appear to be advisable in preventing or lessening the likelihood of its occurrence.

The prevention of cardiac arrest lies not only in the careful selection and preparation of the patient for operation and in the choice and administration of a suitable anaesthetic, but also in the facilities available in the theatre for combating the factors that may lead to cardiac arrest, such as hypoxia, surgical shock and haemorrhage.

It is a *sine qua non* that no patient should be brought to the theatre for a major operation, whose blood has not been chemically studied and any defects rectified as far as his condition permits. Special attention should be paid to the concentration of haemoglobin, which is the oxygen carrier, and to the concentration of protein and electrolytes in the blood. Malnutrition should be corrected and depleted glycogen stores replenished. Existing cardiac and pulmonary conditions should be treated.

Adequate pre-anaesthetic sedation to allay nervousness and suitable medication, such as atropine, to depress vagal function, should be given. During the administration of the anaesthetic the blood pressure recordings and pulse rate should be charted. The laryngotracheal reflexes, which are hyperactive during light anaesthesia, must be depressed by appropriate measures and suboxygenation of even the slightest degree must be avoided. Laryngeal and intratracheal manipulations should be delayed until the patient is well anaesthetized. Finally appropriate measures to correct disturbances in cardiac rhythm during operation must be instituted when necessary.

A suitably equipped theatre should contain (1) apparatus for administration of oxygen, including cuffed endotracheal tubes, cylinders of oxygen and a respirator; (2) a bronchoscopic outfit for aspiration of bronchial secretions; (3) a drug tray containing syringes and needles, a 1 in 1,000 or 1 in 5,000 solution of adrenalin hydrochloride, pronestyl, a two per cent. solution of procaine and a ten per cent. solution of calcium chloride; and (4) a defibrillator.

The use of the cardioscope is becoming increasingly popular and now forms an essential part of theatre equipment. The continuous record of the electrocardiogram on a fluorescent cathode ray oscilloscope making possible the recognition of arrhythmias and changes in conduction may give a timely warning which calls for the use of preventative measures. When cardiac arrest does occur, the cardiograph records its exact nature, whether cardiac standstill or ventricular fibrillation, and the time of its onset, so that valuable time is not lost by delay in recognition and suitable treatment for the

particular type of arrest may be commenced immediately. It is known that the electrocardiograph will record changes when the heart has ceased effective beating, but the pattern is diagnostic of catastrophe and recognizable by any one versed in electrocardiograms. The anaesthetist should be trained to interpret the information given by the cardioscope.

However much we try to prevent it, isolated instances of cardiac arrest will still occur. In certain circumstances, when the anaesthetic induction is stormy, it should be no reproach to the anaesthetist or surgeon if they defer operation to another day.

TREATMENT

The treatment of a patient whose heart has ceased to beat is unequivocal. The essentials are artificial respiration with 100 per cent. oxygen and immediate cardiac massage. The important factor is speed if the patient is to recover with normal cerebral function. Success hangs on early recognition and swift decisive action. The consequences of delay are death of the patient or his survival with cerebral damage — a pathetic figure with defective sight and defective mentality, even leading a vegetative existence.

In a thoracic operation the surgeon can view and palpate the heart to establish the diagnosis beyond doubt; in an abdominal operation he can palpate the aorta. But if evidence of cardiac arrest occurs during induction, the surgeon should act at once. An unnecessary scar on the chest of a living patient is of little consequence when compared with the risk of death by delay in treatment. The first superficial incision to expose the heart will give the answer. Where there is no bleeding there is no circulation and there should be no delay.

The resuscitative measures should be directed to the immediate restoration of oxygen supplies to the tissues, particularly to the brain and heart, and restoration of spontaneous respiratory and cardiac activity. The first aim is the immediate and simultaneous performance of artificial respiration and artificial circulation.

Artificial respiration may be adequately and efficiently maintained with an anaesthetic machine permitting a closed circuit technique, which allows rhythmic ventilation of the lungs with 100 per cent. oxygen by intermittent compression of the breathing bag. A well-fitting mask may serve to commence with, but a cuffed endotracheal tube permits of more efficient pulmonary ventilation and should be inserted as soon as possible. Should there be any obstruction by bronchial secretions, their immediate aspiration is essential so that oxygen can reach the pulmonary alveoli. An unobstructed airway is a necessity. An expert anaesthetist used to such manoeuvres can carry out these procedures and commence pulmonary ventilation in a matter of seconds.
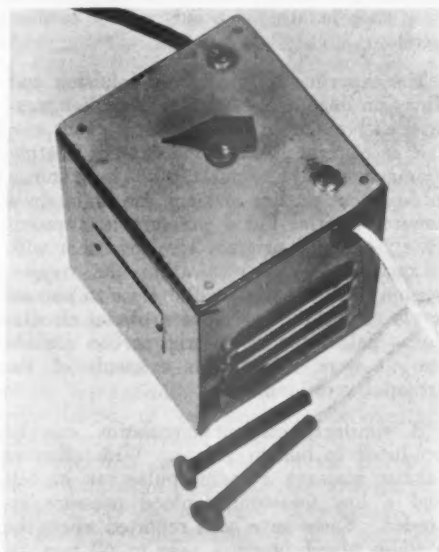

FIG. X. The defibrillator, made by Mr. G. W. Latham of Hallstrom Institute of Cardiology, Royal Prince Alfred Hospital. A 2:1 stepdown transformer with push button for control of duration of shocks, and electrodes for application to the heart. By this means, a 120 volt 50 cycles per second alternating current is applied to the heart for about ½ sec. This may need to be repeated.

Artificial respiration may need to be continued from a few minutes to several hours after the reappearance of cardiac activity, because restoration of spontaneous cardiac action does not necessarily mean the immediate return of respiratory activity.

An artificial circulation can be produced by cardiac massage. Artificial respiration alone is insufficient and is useless unless a

circulation of the oxygenated blood can be effected.

The final measures to be taken to restore automatic circulation will depend on whether the heart is in standstill or in a state of ventricular fibrillation, each of which requires its own particular treatment, but in both the first procedure is massage of the heart at a rate of 50 to 60 per minute, preferably performed through the open chest and intrapericardially. It is generally agreed that massage should commence within three and preferably two minutes. It should be borne in mind that the cortical cells may have suffered partial deprivation of oxygen for some time before the occurrence of cardiac arrest.

The experimental studies of Johnston and Kirby on dogs have shown that cardiac massage will maintain a circulation about one half as efficient as when the heart is beating spontaneously. Ehrenhaft *et alii* have shown in dogs that cardiac massage can maintain a femoral arterial blood pressure at approximately half the original level and that with adequate artificial respiration the oxygen tension can be maintained at close to normal levels during the period of artificial circulation. Values for the arterial carbon dioxide tension were lowered as a result of the artificial ventilation.

A similarly efficient circulation can be produced in human beings. With effective cardiac massage a radial pulse can be felt and a low measurable blood pressure recorded. Cases have been reported where the systolic blood pressure rose to 60 mm. of mercury, but in our four reported cases the pressure did not reach that level. However, sufficient circulation of oxygenated blood can be produced to maintain the integrity of the cortical cells. This fact probably accounts for some reported cases of recovery after long periods of cardiac arrest. Touroff and Adelman reported a case of resuscitation after 40 minutes. How long cardiac massage should be carried out in the face of failure of resuscitation cannot be categorically answered. We suggest a period of one hour as reasonable in most instances.

Technique of cardiac massage

A decision to open the chest is sometimes called for as a diagnostic measure when doubt exists from other signs as to whether the heart has stopped. Certainly it is the most satisfactory means of differentiating between cardiac asystole and ventricular fibrillation in the absence of an electrocardiogram. Once a decision is made to open the chest it should be done with the greatest despatch. Skin preparation, sterile drapes and aseptic technique are discarded in favour of speed. The only instruments necessary are a scalpel and self-retaining retractor. Transthoracic massage through the open pericardium is the only certain way of creating an effective and efficient circulation at once. Johnston and Kirby have demonstrated that direct massage promotes a cardiac output five times that of subdiaphragmatic massage. An intercostal incision should be made from the sternum to the posterior axillary line through the left fourth or fifth intercostal space, with division of the adjacent costal cartilages. There is of course no bleeding. To open the chest and pericardium and commence cardiac massage should not take 20 seconds.

If after a few preliminary compressions of the heart from outside the pericardium, no automatic heart beats can be felt, the pericardium should be widely opened and direct cardiac pumping instituted.

The term "massage" is not quite accurate. The surgeon actually pumps the heart by concentric compressions of the ventricles, a reverse milking process, which reproduces a systolic action of the heart at a rate of between 50 and 60 compressions per minute. Care should be taken to compress the ventricles only and not the auricles so as not to interfere with the diastolic filling of the heart from the large veins in the systemic and pulmonary circulations. Attention to detail is important. Gunn emphasizes the point that compression should be gradual and relaxation abrupt and that short pauses be allowed for ventricular filling during relaxation. The massage should be interrupted at regular intervals for a few seconds to see if spontaneous beats develop. All manipulations should be gentle to avoid injuring the heart. At post-mortem examinations localized petechial haemorrhages due to trauma have been found beneath the epicardium. Hurwitt and Seidenberg have reported a case of rupture of the ventricle due to massage for cardiac arrest in a patient who had a recent infarction.

There is no unanimity as to the optimum rate of compression. Beck and Mautz and Barber and Madden recommend a rate of 40 to 50 per minute, whilst Johnston and Kirby conclude that massage should be carried out at the most rapid rate at which the heart can be compressed and at least 120 times per minute. The suggested rate of betwen 50 and 60 per minute appears to be effective and can be carried out by the operator for some time without his suffering undue fatigue; it also permits of adequate diastolic filling of the heart.

Exposure of the heart through the left side of the chest rather than the right is favoured on account of the easier access to the ventricles, but under certain circumstances when there is one normally functioning lung and one diseased lung, as in Case 4, it is preferable to open the chest on the affected side and so leave the one normally functioning lung uncollapsed for pulmonary ventilation. In the case of tuberculous patients, who have undergone a thoracoplasty and have only one active lung, cardiac massage by the abdominal route is more effective than mere artificial respiration and adrenalin injections as recommended by Bjork.

A slight lowering of the body into the Trendelenberg position will increase the flow of blood o the brain and augment the coronary circulation. Compression of the descending thoracic aorta will have a greater effect by producing a considerable rise in the pressure of blood within the heart and brain. The application of a clamp to the aorta has been recommended but direct pressure by a swab stick held in one hand or direct pressure by the closed fist of an assistant on the abdominal aorta is all that is necessary during the critical period.

Cardiac standstill

When the heart is in standstill or asystole, it may start with the aid of massage alone. After cardiac massage has been carried out for a sufficient time to reduce intra-cardiac pressure and lessen cyanosis, an intra-cardiac injection of two or three cubic centimetres of a 1 in 1,000 solution of adrenalin hydrochloride diluted ten times, as recommended by Beck, will aid in initiating contractions or will increase the strength of feeble heart beats. Evidence is accumulating that an intra-cardiac injection of adrenalin given whilst the heart is at standstill in diastole may lead to ventricular fibrillation. Any such injection should be deferred until cardiac massage has been commenced. After the injection massage is resumed and in most cases forcible rhythmical contractions soon appear. Injection into the left ventricle reaches the coronary circulation quickest. Beck however prefers injection into the right ventricle because it reaches the coronary circulation more diluted and may thereby obviate the danger of ventricular fibrillation. For this reason others favour intra-auricular rather than intra-ventricular injection. Intra-cardiac injections of adrenalin are especially prone to precipitate ventricular fibrillation when trilene, chloroform or cyclopropane have already been administered.

Lahey recommends the injection of adrenalin and procaine in combination, by a single injection of 0.5 cubic centimetre of a 1 in 1,000 solution of adrenalin and 9.5 cubic centimetres of one per cent. procaine.

Kay and Blalock recommend the use of calcium chloride. The calcium ion has a direct effect on the myocardium during standstill, increasing the excitability and contractility of ventricular muscle. It can be used in the treatment of ventricular standstill arising spontaneously or following electrical defibrillation of the heart in ventricular fibrillation. Provided it is injected into the left ventricular cavity, some of the solution can be forced into the coronary arteries by means of cardiac massage. The dosage is two to four cubic centimetres of a ten per cent. solution as a single injection, but it may be repeated after two to three minutes, if considered necessary. Cardiac massage must be continued during this procedure. Blalock suggests that calcium chloride may be more valuable than adrenalin.

Calcium gluconate has also been used in ventricular standstill during surgical procedures on the heart, with resumption of effective cardiac activity, but some contend that it is not as effective as calcium chloride.

It has been recently recommended that after a regular beat has been established noradrenalin should be used to insure that the coronary blood flow, which depends upon the diastolic blood pressure, is maintained. By

this means the oxygen debt of cardiac muscle, which has accumulated during the period of arrest, is liquidated. Although cardiac massage may maintain the peripheral circulation, the blood pressure may be so low as not to be recordable. Nor-adrenalin has a powerful vaso-constrictor action. For use in these emergencies one to two cubic centimetres of a solution containing 20 microgrammes per cubic centimetre are injected into the vena cava where it enters the heart or into an auricle or pulmonary vein. Nor-adrenalin may be a two-edged sword if it causes cerebral vaso-constriction.

In cases where a sudden severe haemorrhage may be a contributing factor to cardiac arrest, but never otherwise, adequate, rapid replacement of the blood lost must be carried out. An intra-arterial blood transfusion is recommended.

When cardiac contractions occur and the circulation has been re-established, the pericardium should be closed with interrupted sutures, haemostasis being effected before wound closure. An intercostal catheter connected to an underwater drainage bottle aids lung expansion while the use of antibiotics protects from infection.

Ventricular fibrillation

Fortunately ventricular fibrillation, which has a far worse prognosis, is less frequently encountered than simple ventricular standstill. It may be a primary condition found on opening the chest or it may develop during massage of the heart for cardiac standstill. Isolated recoveries have followed treatment by cardiac massage, in conjunction with intracardiac injections or topical application of procaine (vide Case 4), but successes have been few. Lampson et alii have described a successful issue after use of this method but more radical treatment is usually necessary. It is now recognized that the only effective means of stopping ventricular fibrillation is by the use of the "defibrillator" (Fig. X), an electrical shock method devised by Beck and Wiggers. A strong current passed through the heart will cause a simultaneous contraction of all the inco-ordinated fibrillating fibres and relaxation follows. The heart is then in standstill and the treatment of this clinical entity should follow. It is recommended that defibrillation should not be attempted until anoxia of the heart has

been overcome. Cardiac massage is still an important preliminary as it reduces dilatation and lessens anoxia of the heart.

The technique now universally employed is that of Beck and Rand. Preliminary massage is carried out to overcome dilatation and anoxia. Five cubic centimetres of two per cent. procaine or 100 to 500 milligrammes of "Pronestyl" are injected into the right ventricle and the shock is then given. If the procedure proves successful, the heart muscle becomes contracted and is brought to a standstill. Thereafter normal rhythm may be induced by cardiac massage. If the method fails, the injection of more procaine and a further shock for a longer period are necessary. When normal rhythm is resumed the heart is sometimes flabby and the heart beats weak. Under these circumstances an injection of adrenalin or calcium chloride is indicated.

Chemical defibrillation by the use of potassium chloride has not proved successful in man.

An automatic respirator may have to be used for some time until spontaneous respiration is established.

In both cardiac standstill and ventricular fibrillation the resumption of the cardiac beat may be transitory at first and the surgeon should therefore be ready to repeat cardiac massage as often as necessary until regular and strong contractions are permanently re-established.

In few other conditions are the scales so delicately balanced as to be tipped easily either towards life or towards death. Attention to detail and efficient therapeutic measures speedily carried out are rewarded with gratifying results. When life returns under the stimulus of succesful cardiac massage, the operator has a pleasant feeling of achievement. By a simple procedure he ignites that vital spark which sets in motion complex physiological processes which enable the apparently dead to rise and continue their journey through life.

SUMMARY

The subject of cardiac arrest is discussed with mention of primary and secondary types.

The parts played by the various intravenous anaesthetic agents in the causation of

cardiac arrest during induction of anaesthesia, is discussed.

An attempt is made to list the various causes of cardiac arrest. Certain observations were made on patients who had normal cardiac and respiratory function, and who were undergoing operation. These observations included direct arterial blood pressure, right auricular pressure and the electrocardiogram, particularly during the induction of anaesthesia.

In a group of patients undergoing thoracotomies for respiratory or cardiac disease, electrocardiographic studies only were made.

Four cases of cardiac arrest are reported in which the patients were treated by cardiac massage. There were three recoveries.

Prophylaxis is mentioned.

The surgical and therapeutic measures to meet the emergency of cardiac standstill or ventricular fibrillation are detailed.

Direct cardiac massage through the open thorax and pericardium within two minutes of the catastrophe is advocated. The necessity for speed is stressed.

ACKNOWLEDGEMENTS

We are indebted to Dr. Kempson Maddox for helpful suggestions and criticism of this paper and to Mr. Reg. Johnson of the Department of Medical Film Research, Royal Prince Alfred Hospital, for all the photography.

ADDENDUM

Since the presentation of the above paper we have successfully treated another patient suffering from cardiac arrest.

A male child aged three years was admitted to the Royal Prince Alfred Hospital with congestive cardiac failure for investigation and treatment. A liver biopsy under general anaesthesia was recommended.

The anaesthetic administered was ethyl-chloride and ether applied on an open mask. It was accompanied by the continuous administration of oxygen.

Four minutes from the application of the anaesthetic the respirations became depressed and the colour of the boy grew cyanotic and cardiac arrest at once ensued. Immediate intubation was performed, 100 per cent. oxygen administered, and trans-thoracic cardiac massage commenced. The heart was found to be dilated and in asystole. Within two to three minutes normal cardiac rhythm was restored.

The child made an uneventful recovery with normal vision and without evidence of cerebral damage.

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THE PRESENT RATIONAL TREATMENT OF PORTAL HYPERTENSION

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PORTAL hypertension is essentially a state of congestion of the portal venous system due to mechanical obstruction of the portal venous outflow. The portal venous pressure is dependent upon the *vis a tergo* of the systemic blood pressure which propels the portal blood through the splanchnic capillary bed; against this is set the resistance to the outflow of portal blood by the liver "sponge" and portal-systemic communications. It follows therefore that the portal venous pressure can never exceed the mean systemic arterial pressure. It is important to regard portal hypertension as a mechanical fault in which a dam has been thrown across the portal blood stream, thereby hindering the onward portal flow. As a result, a head of pressure is raised behind the dam to help overcome the unnatural resistance. From this concept the symptomatology may be readily deduced, and a rational form of treatment evolved.
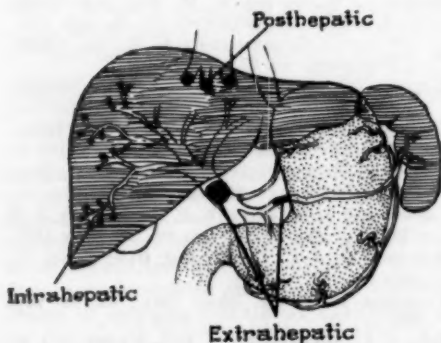

FIG. 1. Sites of possible portal venous obstruction.

AETIOLOGY

The portal circulation is unique in that it commences in capillaries and ends in capillaries. Therefore, theoretically the portal pressure could be raised by increased flow and pressure through the mesenteric capillary bed; in actual fact this does not occur. Instead, increased resistance to the outflow develops. This may occur either in the main portal vein itself or in the portal tracts within the liver, or in the liver sinusoids, or in the outflow from the liver sponge, viz. the hepatic veins. Thus there is

> Prehepatic obstruction
>
> Intrahepatic obstruction
>
> Posthepatic obstruction.

Prehepatic obstruction

This represents obstruction in the portal vein itself. Portal vein thrombosis may occur as an acute incident producing a serious surgical emergency which is usually fatal. But it is believed to occur more commonly as a quiet ante-natal or neonatal condition. It is thought by some that the normal process of obliteration of the umbilical vein and ductus venosus may extend and obliterate the portal vein itself or that the portal vein may fail to develop. Another possible cause of portal vein obliteration is a quiet thrombophlebitis developing soon after birth from an omphelitis. The thrombophlebitis spreads back along the umbilical vein. Consequently an atretic fibrous cord may be all that is left to suggest the existence of a previous portal vein, or else all vestiges of it are non-existent. In its place a mass of thin-walled collateral veins develop—the so-called cavernous transformation. Under these circumstances the liver parenchyma will be normal histologically, and therefore liver function will be normal. Occasionally the portal vein agenesis may extend into the liver and the portal venules will be absent on the portal tracts. Another and rare form of prehepatic obstruction is that due to stricture of the portal vein. Milnes Walker records such a case demonstrated by portal venography (1953) and Whipple (1945) records 4 such cases. Mahoney and Hogg (1950) record two such cases treated surgically.

Patients suffering from extrahepatic portal obstruction are usually young children whose symptoms of haematemesis develop early, often before the age of 5 years, but the onset of symptoms may be delayed till in the late teens. These children are said to suffer from Banti's disease or the Banti's syndrome, i.e. splenomegaly, anaemia and neutropenia, with oesophageal varices. It would, however, be very much better if the term Banti's disease could be deleted from the literature and the term Extrahepatic Portal Vein Obstruction, or Extrahepatic Portal Hypertension be substituted. Because the idea still persists that the splenomegaly and splenic fibrosis may be primary and that subsequently this condition may spread to the liver and produce a secondary portal cirrhosis. It is on this fallacious reasoning that splenectomy has been justified in these cases. In actual fact, patients with an extrahepatic portal vein obstruction have normal livers and never develop portal cirrhosis unless they should happen to suffer from necrotic or metabolic liver disease quite unrelated to the portal vein block. Splenectomy alone, therefore, is never justified in these patients. Spleno-renal anastomosis should always be done at the time of the removal of the spleen. Indeed a splenectomy is a surgical crime, because thereby the splenic vein is destroyed, and with it is destroyed the only chance of doing an adequate venous shunt. Furthermore, splenectomy is quite ineffective in preventing further haemorrhage from the oesophago-gastric varices, except in the rare instance of a thrombosis of the splenic vein itself with a normal portal vein.

Because a normal liver is present in these patients with extrahepatic portal vein block, normal liver function is found. Therefore, ascites should not develop. However, there is a set of circumstances under which ascites may occur, viz. following a severe haematemesis producing a fall in the blood proteins, and particularly in the serum albumin. Under these circumstances, with a serum albumin below 3 mg. per cent. ascites may appear but will resorb when the blood proteins return to normal levels.

The prognosis is bad in extrahepatic portal hypertension unless a satisfactory venous shunt can be accomplished.

Intrahepatic block

Blockage of the portal venules in the portal tracts is usually secondary to cirrhosis of the liver and therefore acquired. The cause of cirrhosis of the liver is debatable, and indeed there are multiple causes. Some cases are undoubtedly secondary to some form of previous hepatitis which may or may not have been recognized clinically at the time. At least fifty per cent. of patients suffering from portal hypertension have no previous history of clinical hepatitis. The part that alcohol plays in the origin of Laennec's cirrhosis is uncertain — by itself it appears on experimental grounds to be unable to cause anything other than fatty infiltration of the liver, but it does appear to potentiate the action of such liver poisons as carbontetrachloride or phosphorus in a very striking manner. A prolonged deficient diet and inadequate vitamin intake are certainly important causes of cirrhosis in the chronic alcoholic. It therefore follows that intrahepatic portal obstruction is more likely to be met with in adult life. Although fully developed Laennec's cirrhosis may be seen in children before puberty, this is uncommon. In countries where schistosomiasis is endemic hepatic cirrhosis due to this cause is frequent and quite young children may present with a severe portal hypertension as a result.

Posthepatic block

This is a relatively uncommon condition involving some process blocking the hepatic veins thereby producing the Budd-Chiari syndrome. It is not uncommonly associated with obstruction of the inferior vena cava at the level of the entrance of the hepatic veins. One of the commonest causes of this condition is neoplasm. It is not intended to discuss this further as it is outside the ordinary concept of portal hypertension.

Milnes Walker (1952) has pointed out that there are three degrees of portal cirrhosis:

(i) Involving the portal tracts only.

(ii) Involving the portal tracts and spreading between the liver lobules to join up adjacent portal tracts.

(iii) Involving portal tracts and spreading diffusely to involve the central lobular vein and generally disorganizing the whole liver structure.

All these types may be associated with hypertension but types (ii) and (iii) are liable to have, as well, moderate to severe degree of liver failure. It therefore follows that type (i) should suffer from haemetemesis alone, whereas types (ii) and (iii) are likely to have haemetemeses plus ascites.
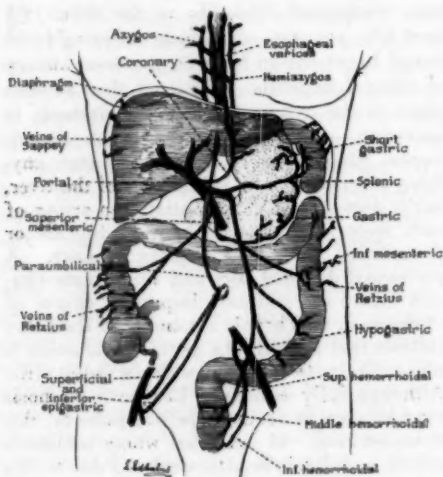

FIG. II. Diagram of portal systemic venous anastomoses in portal vein obstruction.

The normal portal venous pressure is 80-150 mm. of water. In portal cirrhosis this may rise up to 500 mm. of water, though a usual average pressure producing symptoms in 350 mm. of water. As a result of this abnormally high pressure, the thin-walled poorly supported mesentric veins dilate and become tortuous, and the various communications between the systemic and portal systems open up. These communications, or so-called collaterals, are divisible into three groups:

(1) Those around cardia of stomach and oesophagus joining with the azygos system.

(2) Those around the rectum joining with the pudic veins.

(3) Those in retroperitoneal tissue joining with lumbar veins, and those in the anterior abdominal wall joining with epigastric veins.

As well as these communications normally present, numerous very vascular adhesions develop between the liver and abdominal wall, and between the great omentum and the abdominal wall.

By these means nature tries to so open up the collateral bed, that the effect of the "dam" is reduced and the hypertension falls as a result. Indeed, one sees cases in which some hypertension is known to have existed in the patient in the past as evidenced by a history of haematemesis, but in whom, in the course of time, the pressure has fallen due to the opening up of these collaterals. One case will serve to illustrate this point:

Mrs. S., aged 50 years. Twenty years previously had two episodes of haemetemesis. Subsequently she was admitted for another abdominal complaint and was found to have an enlarged spleen. Barium swallow and oesophagoscopy revealed typical varices in the oesophagus, but these were well covered by normal mucous membrane. At the subsequent abdominal operation, the portal pressure was measured and found to be 120 mm. of water, well within normal limits; yet 20 years ago it must have been raised. The liver showed a typical Stage I cirrhosis.

The danger of portal hypertension lies in this very effort of nature to establish an efficient collateral circulation. Little harm results from the retroperitoneal collaterals, or the rectal ones. Contrary to textbook accounts, it is rare for haemorrhoids to develop in portal hypertension, though one does see dilatation of the superior haemorrhoidal veins; it is rarer still for them to bleed. But the anastomoses between the left gastric vein and the azygos system become very large and represent the "Sword of Damocles" in this condition; it is through them that portal hypertension kills.

Gross thin-walled varicosities may appear submucosally in the fundus of the stomach and in the whole length of the oesophagus although they are more common in the lower one-third. The mucosa over these may become ulcerated and then the varix ruptures and profuse haematemesis of melaena results. It is possible that the negative pressure in the thorax acting on these very poorly supported, thin-walled, veins is a factor in making these collaterals so dangerous. Certainly co-existing oesophagitis and ulceration are most important.

Oesophageal varices in themselves are not dangerous, but varices plus hypertension are potentially lethal. If it is possible to tide the patient over till nature has reduced the hypertension by opening up an adequate collateral bed, or else, if it is possible to reduce the hypertension by operation, then the patient will be secure.

FIG. III. X-ray of barium swallow showing marked oesophageal varices in lower third of oesophagus.

SYMPTOMATOLOGY

The leading symptom of portal hypertension is haematemesis. Ascites is a symptom of liver dysfunction. It may be associated with portal hypertension, but can occur without it. On the other hand, haematemesis does not occur without hypertension. On clinical examination of a patient with a Laennec type of cirrhosis there is an enlarged firm spleen, unless it has unfortunately been previously removed. The liver is frequently enlarged and firm, but may be normal, or shrunken in size. Ascites may be present, but frequently is not. There may be evidence of enlarged veins in the abdominal wall and in the lateral thoracic regions.

Certain investigations are essential. A full blood count is required. Some of these cases have a pancytopenia or a leucopenia, possibly due to hyper spleenism. The E.S.R. should be normal. The blood proteins must be known and the A/G ratio should not be reversed. This is one of the most useful tests of liver function. If the proteins are normal, then the liver function is usually satisfactory. The blood prothrombin level is also a good gauge of whether liver function is adequate. The serum bilirubin should be normal. The Brom sulfaphthalein retention should be less than 5 per cent. after 30 minutes.

A barium swallow will usually show up oesophageal varices, without which, a diagnosis of portal hypertension should not be made without confirmation by direct measurement. There are, however, cases of proven portal hypertension who have suffered a massive haematemesis, in whom no varices can be demonstrated. The source of bleeding in these patients is not known, but it may be an acute gastric erosion. If there is doubt, then an oesophagoscopy and possibly a gastroscopy should be performed. Oesophagoscopy is useful, because it gives an accurate picture of the extent of the varices and the state of the oesophageal mucosa overlying them. Where hypertension is present, and haematemesis likely, the mucosa is thinned out over the varices, and small areas of ulceration may be seen; oozing of blood may occur from the trauma of the instrument. On the other hand, if the hypertension is not present, the varices, though readily visible, will be covered with normal thick mucosa, and no bleeding occurs from the passage of the instrument.

If the spleen is still present, great help is to be derived from a splenogram. This is an X-ray examination whereby the portal vein and its tributaries may be outlined by a radio-opaque dye. Thirty cc. of 70 per cent.

diodone is injected by per-cutaneous puncture into the enlarged spleen and an exposure made immediately upon the completion of the injection. By this means, the patency or otherwise of the splenic portal veins may be demonstrated, and the flow of blood up the oesophageal varices shown. (M. Walker, *et alii*, 1953.) This procedure is particularly useful in demonstrating the site of block in cases of extrahepatic block, and saves a fruitless exploration of the hepatic pedicle in such patients.

FIG. IV. Splenogram of a patient with portal hypertension due to Laennec's cirrhosis. Note marked oesophageal varices demonstrated by diodrast.

TREATMENT

If liver function is subnormal, as shown by the presence of ascites, or poor liver function tests, then it is wise to treat the case medically by high protein, high carbohydrate, high vitamin diet, with low salt intake and mercurial diuretics. Ascites by itself is not an indication for surgical treatment. If liver function is normal, then the object of treatment is to relieve the tension on the thin-walled oesophageal and gastric varices. If possible this should be achieved by a venous shunt. Credit for developing the venous shunt as a means of treating portal hypertension

must go to Blakemore (1945) and Whipple (1945). The best shunt to make is a porto-caval one if the portal vein is patent. If not, the second best is a spleno-renal anastomosis. Any shunts other than these are useless, being too small and too unlikely to remain patent. More collaterals will be destroyed in creating these small shunts than will be compensated for by them. If no adequate shunt is possible, as in cases of cavernous transformation, in which the spleen has unfortunately been removed, then the only manoeuvre left is to interrupt the flow of blood through the dangerous gastric or oesophageal varices. This may be accomplished by the gastric transection suggested by Tanner (1950), using the transthoracic route, or by oesophageal transection, or by the Allison (1950) manoeuvre of dividing all feeding veins to the oesophagus and "ring barking" it. Crile (1950) and ohers have used oesophogotomy and direct ligation of the varices by trans-fixation. These procedures will obviate the danger temporarily but no doubt revascularization will occur across the suture lines; but they may tide the patient over sufficiently long for an adequate collateral bed to develop and thus reduce the hypertension naturally. The young patients with extrahepatic portal block have a very bad prognosis and therefore any rational procedure is justified. If the spleen is still present, then a spleno-renal anastomosis end to side, with splenectomy, is the ideal procedure; but in so many of these cases splenectomy has already been performed and then some form of obliterating procedure on the varices must be done.

Fig. VI (a), (b) shows X-rays of barium swallow in a child of 8 years of age with congenital obliteration of the portal vein and portal hypertension in whom the spleen had already been removed. Fig. VI (a) shows marked oesophageal varices before operation. Fig. VI (b) shows disappearance of varices following an operation at which the stomach was transected and the oesophagus opened and the varices ligated. The barium swallow was taken six months after operation. In these enlightened days it should be considered a surgical crime to perform a splenectomy for portal hypertension of extrahepatic origin unless a spleno-renal anastomosis is done at the same time.
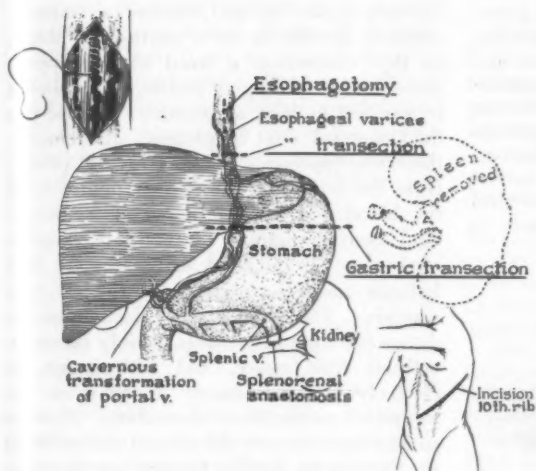
FIG. V.   Four procedures available to treat portal hypertension due to extra hepatic block.

Porto-caval anastomosis

The best approach for this is a thoraco-abdominal incision through the bed of the 9th rib, and incising the diaphragm back to the right coronary ligament. By this means the liver may be dislocated into the thorax thus opening up the subhepatic area. The portal vein is then approached from the side, "sneaking in" underneath the numerous vascular adhesions found from the ventral approach. It is usually possible to isolate and ligate the right and left branches of the portal vein, thus giving extra security to the ligatures and a greater length of portal vein to work with. The inferior vena cava is readily defined and its anterolateral surface cleared for anastomosis. The anastomosis is made end to side, using the usual everting continuous mattress sutures. Once the clamps are released, the portal blood may be seen swirling into the inferior vena cava, and if the portal pressure is now taken it will be found to have fallen to a reasonable figure, e.g. 350 mm. of water down to 220 mm. of water.

Post-operatively, splenography may again be used to demonstrate the patency of the anastomosis, and the reduction in the portal hypertension. When the dye is injected into the spleen after a successful shunt, it will be seen to pass rapidly into the inferior vena cava and the radicals of the portal vein such as the left gastric and gastro-epiploic veins will no longer show up. The oesophageal varices will not be outlined, showing that the direction of flow of portal blood has now been reversed. Therefore, by deduction, the portal hypertension has been reduced and the strain on the oesophageal varices relieved, and the danger of rupture averted.

The danger of porto-caval shunt lies in the possible onset of hepatic coma in the post-operative period due to poor liver function. But this does not occur where the liver function tests have been normal pre-operatively. It is, however, a very real danger in case of lowered liver function as occurs in Stage II and Stage III liver cirrhosis.

(a)   (b)

FIG. VI.   (a) The barium swallow before operation demonstrating marked oesophageal varices.
(b) The barium swallow 6 months after operation showing disappearance of varices.

An interesting feature of a successful porto-caval shunt is that the enlarged spleen frequently shrinks to half its former size and becomes less firm. Also where a leucopenia has existed before operation, this may correct itself with the relief of the splenic congestion. Learmonth's contention (1951) that splenectomy is required in these cases to correct hyperspleenism after a successful porto-caval shunt has not been found to be necessary in many cases.
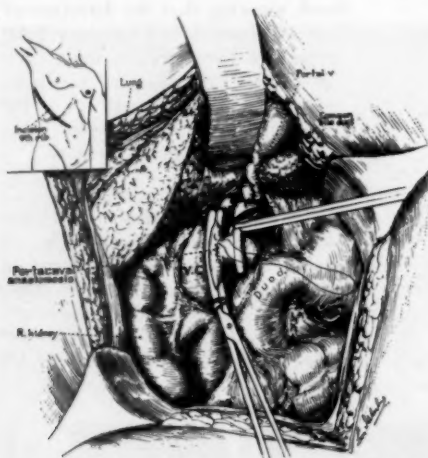

FIG. VII. Technique of porto-caval anastomosis.

COMPLICATED CASES OF PORTAL HYPERTENSION

Thus far uncomplicated cases of portal hypertension have been considered and their management is relatively straightforward. But the really difficult patients are those who have a portal hypertension plus liver failure. They usually present with a haematemesis, have massive ascites and probably a grossly enlarged liver and jaundice. The emergency treatment is to deal with these haematemesis by transfusion, and control the bleeding point by a tamponading oesophageal tube of the Sengstaken type (1950). If bleeding will not cease or recurs shortly, an emergency direct attack on the oesophageal varices by a transfixion or a transection procedure may be necessary. A vigorous attack must then be made on the ascites and poor liver function, in an effort to tide them over until their liver

function picks up and reaches a stage at which it is safe to do a porto-caval shunt. In this connection a word of warning is necessary about tapping ascites. This should be avoided as long as possible, because this fluid contains a lot of protein. Its removal, therefore, represents the loss of this protein from the body and the further depletion of the blood and body proteins as the ascitic fluid reaccumulates. Furthermore, massive removal of ascitic fluid will upset the fluid balance and electrolytes of the body very severely. The fluid which reaccumulates is drawn from the body fluids, mainly the extracellular fluid space, and with it go the electrolytes approximately in the same concentration as the blood electrolytes. Thus the patient may become dehydrated and deficient in electrolytes purely because he has been drained into a useless pool in his peritoneum. It is probable that many so-called hepatic comas following abdominal paracentesis represent nothing more nor less than gross fluid and electrolyte disturbance.

In the treatment of hepatic coma two recent developments are of interest. Walshe (1953) has shown that administrating glutaminic acid either by mouth or intravenously may have a beneficial effect though others have not found this specific. The other drug worth using is aureomycin at the rate of 3 grams a day given intravenously. The efficacy of this drug suggests that liver failure may be due to a low grade hepatitis.

In the treatment of the difficult cases of combined portal hypertension and liver failure Rienhoff (1951) and others have advocated hepatic artery ligation. Suffice it to say that so far this procedure has not been generally accepted, and is still under trial.

CONCLUSIONS

1. Two types of cases of portal hypertension require surgical treatment:

   (a) The extrahepatic portal block.

   (b) The intrahepatic portal block.

2. The extrahepatic portal block is usually congenital; the most frequent anatomical defect is a cavernous transformation of the portal vein. Such cases commonly

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present in young children with a pure portal hypertension producing haematemesis. Their livers are normal anatomically and functionally. These cases should be treated by a spleno-renal anastomosis. If that is not possible due to the loss of the spleen, then direct attack on the varices is indicated by a transection or a transfixing procedure.

3. Intrahepatic portal cirrhosis is usually of the Laennec variety. Here a good portal vein usually exists, and provided adequate liver function is present, a porto-caval, end to side anastomosis is the treatment of choice.

4. Ascites and poor liver function are contra-indications to any shunt procedures until the liver function is back to normal or near normal; but if haematemesis is likely to prove fatal in these cases, then a direct attack on the varices is indicated as a life-saving procedure, with the idea of doing a shunt later.

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RADICAL PANCREATO-DUODENECTOMY FOR AMPULLARY CARCINOMA

A CASE REPORT

By Howard H. Eddey

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OBSTRUCTION of the lower end of the common bile duct by carcinoma is due most often to a carcinoma commencing in a small pancreatic duct in the head of the pancreas and secondarily involving the common bile duct. Much less frequently the carcinoma commences in the region of the ampulla of Vater, the possible sites of origin being the duodenal mucosa covering the duodenal papilla, the lining of a true ampulla of Vater, the terminal common bile duct, the terminal pancreatic duct or very rarely in Brunner's glands or aberrant pancreatic tissue in the duodenal wall.

Few cases of carcinoma of the head of the pancreas are found at operation to be suitable for radical pancreato-duodenectomy and even if this operation is technically possible, the operative mortality is not inconsiderable and the patients who survive longer than one year are distressingly few. This operation is the one of choice for a carcinoma of the ampulla, provided the patient's general condition is satisfactory, for the operative mortality is reasonable and the chance of cure good.

The following case report is of a patient who was operated upon for obstructive jaundice, the cause of which was not recognized until a second operation was performed for persisting obstruction of the lower end of the common bile duct.

CASE REPORT

Mr. S.N., aged 48, was referred to the writer by Dr. H. M. Webber of Albury, who first saw this patient on 1st May, 1953, when he presented with painless jaundice, nausea and weakness. He was regarded as suffering from catarrhal jaundice, but as the jaundice did not subside, he was admitted to the Albury Base Hospital on 15th June, where investigations revealed the jaundice to be obstructive in origin. Operation was performed by Dr. Webber on 22nd June and a distended gall-bladder and common bile duct were found. The pancreas and stomach appeared normal. The gall-bladder was

aspirated and normal bile was seen to be present, but no calculi could be palpated. The common bile duct was opened and a small calculus 3 mm. in diameter removed. A common duct probe was passed into the duodenum and the operation was completed by the insertion of a T-tube into the common duct.

The jaundice subsided slowly after this operation and the faeces assumed a normal colour immediately after operation. However routine cholangiograms showed that no dye passed into the duodenum and the patient could not tolerate clamping the T-tube for longer than two hours.

The patient was admitted to the Royal Melbourne Hospital on 12th August, 1953. On admission, he was in good general condition and the T-tube was draining 24 ounces of bile each day. No clinical jaundice could be detected and the faeces were of normal colour.

The following investigations were carried out:—

1. Blood examination:

   Haemoglobin — 112 per cent. (14.8 gms.)
   — 16.5 gms. per 100 ccs.

   Leucocytes — 6,000 per cmm.

   Films — The erythrocytes, reticulocytes and platelets were normal. The differential count showed a monocytosis.

2. Liver function tests:

   Van de Bergh reaction — Delayed positive.

   Units of bilirubin — 6.

   Fouchet test — Positive.

   Serum protein, total — 7.2 per cent. (17 m. Eq/L.).

   —— Albumen — 2.7 per cent.

   —— Globulin — 4.5 per cent.

   Alkaline Phosphatase — 15 units.

   Prothrombin efficiency — 60 per cent.

   Cephalin flocculation — Negative.

3. Casoni test:

   The immediate and delayed reactions were negative.

4. X-ray examination of the common bile duct (Fig. I).

Opaque medium injected down the T-tube into the common bile duct did not enter the duodenum. The common bile duct was dilated and there was a

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rounded filling defect at its lower end, suggesting a possible calculus impacted in the lower end of the common bile duct. The hepatic ducts were normal.

FIG. I. Photograph of the cholangiogram demonstrating the filling defect at the lower end of the common bile duct.

Arrangements were made for an occult blood examination, but unfortunately this investigation was not carried out before operation.

A pre-operative diagnosis of obstruction of the lower end of the common bile duct due either to an impacted calculus or to a carcinoma of the ampulla of Vater was made.

Operation

Operation was carried out on 19th August, seven days after admission of the patient to hospital. The anaesthetic consisted of pentothal, flaxedil, nitrous oxide and oxygen administered by Dr. V. Lines. Continuous intravenous saline therapy was instituted and two pints of blood were given during the operation. At the end of the operation, the patient's systolic blood pressure was 130 mm. of mercury, the pulse rate 112 per minute and the colour and respirations good.

Operative findings.—The gall-bladder was moderately dilated, but otherwise normal, and the common bile duct was grossly dilated. A small hard mass could be palpated at the termination of the common bile duct and to inspect this mass the duodenum was mobilized and incised opposite the duodenal papilla. A small pinkish mass 0.5 cm. in diameter could then be seen involving the duodenal papilla and the ampulla of Vater, and this mass appeared to be ulcerating into the duodenum. There was no involvement of the associated lymph nodes. A small

piece of the ulcerated area was examined by frozen section, but this revealed some tubules only and one "suspicious area." An operative diagnosis of carcinoma of the ampulla was made and a partial pancreato-duodenectomy was carried out.

Operative technique. — The T-tube was removed from the common bile duct. The duodenum and the head of the pancreas were mobilized, the pancreas was divided through the neck after freeing the superior mesenteric and portal veins, the stomach divided through the pyloric antrum and the duodenum divided at the duodeno-jejunal flexure. The common bile duct was divided in the gastro-hepatic omentum and the gall-bladder was removed. The first part of the jejunum was mobilized and brought up behind the superior mesenteric vein. The cut end of the jejunum was oversewn and end-to-side anastomoses were carried out in turn between the common bile duct, pancreatic duct, cut edge of the stomach and the jejunum. The wound was closed with a drain tube into the hepato-renal pouch.
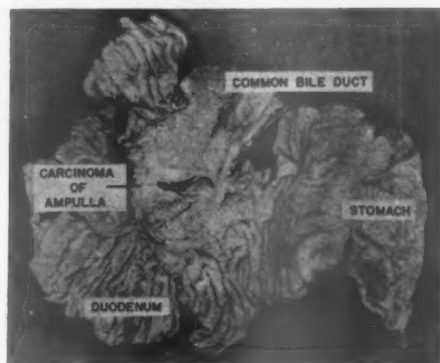

FIG. II. Photograph of the specimen removed at operation illustrating the carcinoma at the termination of the dilated common bile duct.

Pathological report (Dr. J. D. Hicks).—"Macroscopically: The specimen consists of the distal 3 cm. of the stomach and 15 cm. of duodenum. There is a thick walled stump of the common bile duct measuring 5 cm. in length and which is dilated to three times its normal size. Encircling the ampulla of Vater itself is a pinkish papillary mass 0.5 cm. in length. Also attached to the specimen is 5 cm. of the head of the pancreas. Included with the specimen, there is a normal looking gall-bladder 6 cm. in length.

"Microscopically: There are irregular glands extending through the submucosa of the duodenum and down between the bundles of the muscle coat to the pancreatic tissue outside. Some irregular tubules are seen in the mucosal surface, which is ulcerated and inflamed. The gall-bladder shows a rather thick mucosa with some round cell infiltration.

"Diagnosis: Adenocarcinoma of the ampulla of Vater. Chronic Cholecystitis." (Fig. II shows the specimen which was removed.)

Post-operative progress.—The patient had a smooth convalescence and was discharged to a convalescent hospital sixteen days after operation. Some bile drained through the tube for forty-eight hours and a moderately severe wound infection developed and pus was discharged on the 12th post-operative day. A pancreatic fistula did not develop. The Van den Bergh reaction and Fouchet test became negative and the serum biluribin dropped to one unit.

The patient was readmitted from the convalescent hospital eighteen days later because of upper abdominal pain. Examination revealed thickening and redness of the upper abdominal wounds, but the condition soon subsided following antibiotic treatment. The patient was finally discharged from the Royal Melbourne Hospital one week later and he returned to his home in Albury. He has remained well ever since.

COMMENT

The course of this patient's illness illustrates some points in relation to carcinoma of the ampulla of Vater.

1. The presence of a small calculus in the common bile duct of a jaundiced patient, does not contra-indicate the presence of a carcinoma of the ampulla.

2. Operative diagnosis of a carcinoma of the ampulla is difficult. If a common duct probe cannot be passed into the duodenum at operation or if obstruction persists after exploration and drainage of the common bile duct, the duodenum must be opened and the region of the ampulla inspected.

3. If a small hard lump is felt in the ampulla and this lump is not a calculus, the duodenum should be opened and the lump examined. If necessary, a frozen section biopsy should be undertaken, although the result of the examination may be inconclusive.

4. The operation of radical pancreato-duodenectomy for carcinoma of the ampulla is tedious, but not difficult since the mobilization of the superior mesenteric and portal veins, which is the only difficult part of the operation, is carried out in an area whose anatomy is undisturbed.

THE SEDIMENTATION OF HYDATID SCOLICES

By S. C. Fitzpatrick

Hamilton

ONE of the fundamental technical problems in the surgical cure of the unruptured hydatid cyst is the removal of the cyst without the spilling of hydatid elements, scolices and brood capsules into the host tissues.

There are three possible methods of avoiding this spilling:—

1. Removal of the internal or the whole thickness of the adventitia with the cyst intact.

2. Incision of the adventitia and extrusion of the intact cyst.

3. Partial aspiration of the cyst, incision of the adventitia, and removal of the partially emptied cyst without spilling any of its contents.

All these methods have been used in the past, either by intention or accident. The first method is practicable where the adventitia is sufficiently well developed and where large blood vessels or other important structures do not form part of the wall. This technique is more often possible with lung cysts than with liver cysts. The second method again is more often applicable to lung cysts. Barrett and Thomas (1952) removed intact pulmonary cysts with success in 17 out of 21 attempts. This total extrusion is aided by inflation of the lung by the anaesthetist and the transmitted pulsations from the cardio-vascular system. These aids are absent in removing cysts from solid organs such as the liver and spleen. Extrusion from these solid organs is practicable where the cyst is near the surface of the more easily accessible ventral and inferior surfaces. Natural extrusion has been observed into both pleural and peritoneal cavities.

The third method has been used for many years but without complete understanding of all the factors necessary for success. Either partial or almost complete emptying of the cyst is carried out by using a small or large bore needle, or trocar and cannula, with or without suction bell to catch the small amount of fluid under pressure which escapes at the initial insertion. Barrett and Thomas (1952) record "six patients out of less than fifty" treated by preliminary aspiration who later developed daughter cysts. From my own records, 7 out of 97 patients with pulmonary cysts developed daughter cysts subsequently. Five of these had their primary cysts operated upon elsewhere and no information was available as to whether the cysts were simple or as to the method used. The other two were ruptured before operation. No recurrence has been met with by me after the use of partial aspiration followed by lifting out of the remaining cyst wall intact.

RATE OF SEDIMENTATION OF HYDATID SCOLICES

It has not been satisfactorily explained why recurrence followed in such a small percentage of cases treated by this method. It appears that one factor which may not have received sufficient attention is the rate and completeness of sedimentation of the hydatid scolices. It can be shown that the scolices tend to sediment quite rapidly if given suitable conditions of rest. A transparent plastic container, rectangular in cross section, 20 mm. by 5 mm., and 90 mm. high, was filled with freshly aspirated hydatid fluid from a simple cyst. All the suspended scolices were found to sediment in sixty seconds (Fig. I). It was observed that slight variations occur between fluids from different cysts but the actual variation in time is not more than a few seconds. It is natural to ask whether the scolices vary in weight and if some remain in the fluid even after a preliminary resting period. Also there might be scolices which

have a very slight attachment to the germinal membrane, are about to break free, and are likely to be detached by the outward flow of the fluid when the cyst is first punctured. We have carried out the following tests:— Cysts in sheep and in bovine lungs, varying in diameter from 3 to 10 cm. in diameter, were allowed to remain at rest for three minutes. Each was punctured with a needle and the cyst fluid allowed to rush out until it stopped flowing. The needle had a diameter of 2 mm., B.S.W.G. No. 14, and was inserted to a depth of one cm. into the uppermost part of the cyst. Both the fluid drawn off and the residual fluid in the cyst, was examined macroscopically and microscopically for the presence of scolices.

Provided the internal surface of the germinal membrane was not accidentally touched by the point of the needle, no scolices were found in the run-off fluid. Impact of the needle or cannula on the inside of the germinal membrane detached scolices in some tests. When sedimented all the scolices occupied a depth of .5 mm. to 1 mm. in the bottom of the test container. Scolices appeared to be floating within intact cysts but agitation or alteration of the position of the cyst will show that these scolices are still attached to the germinal membrane and are not freely suspended. Given a period of sufficient preliminary rest and avoiding subsequent disturbance, the possibility of any scolices being present in the fluid which escapes outside the needle when the needle is first inserted at the highest part or zenith of the cyst is probably negligible.

The conditions of successful removal of a cyst by aspiration are:—

1. Controlled respiration to eliminate pulmonary movement.

2. A period of rest of about two minutes after exposure of the adventitial wall sufficient for the scolices to settle at the bottom of the cyst.

3. Aspiration of sufficient fluid by a small bore needle or trocar and cannula inserted at the highest point of the cyst, to lower the intracystic pressure below that of the atmospheric pressure, and to reduce the weight of the residual fluid below that which would cause the cyst wall to tear during lifting out of the cyst wall.

4. Avoidance of puncture or disturbance of the cyst wall at any other point.

5. Lifting the partially emptied cyst out of the adventitial cavity without tearing the cyst wall.

While all the factors which govern the survival of the scolices which have been accidentally introduced into the host tissues during operation are still not completely clear, it is evident that the rate and completeness of sedimentation of the scolices play an important part in the success or failure of surgical treatment of hydatid cysts.

This factor is fundamental when considering the best method of preventing contamination of the host tissues with hydatid elements during the removal of the cyst. The choice of the method of dealing with the unruptured cyst will depend upon the local conditions found during the operation.

SUMMARY

All suspended scolices in an unruptured hydatid cyst at rest will sediment within sixty seconds.

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TRAUMATIC RUPTURE OF THE SPLEEN
A REVIEW OF EIGHTY-FIVE CASES

By NEIL JOHNSON

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IN this modern age when man's lust for speed is entering into every phase of our daily life, it is inevitable that injury to the human body must occur with increasing frequency. For this reason the subject of traumatic surgery is one of ever-growing importance and it is essential that our concept of the clinical picture, displayed by an individual following the receipt of trauma, be accurate. Whilst the clinical syndromes following peripheral injury are relatively simple and easily understood, those following injury to the trunk are more complex and correspondingly more difficult to elucidate. Particularly do these remarks apply to the subject of traumatic rupture of the spleen.

The current teaching on the subject of splenic rupture is still influenced by statements made by authors at the turn of the century and accepted without question since that time. Typical of these statements is that of Berger who investigated a series of cases in 1902. He stated that 50 per cent. of cases of splenic injury, if untreated, will die in the hour following the occurrence of trauma and that 95 per cent. of the cases will die within the first twenty-four hours. Thus rupture of the spleen is considered to be a lesion associated with moderate to severe shock and obvious abdominal signs. Whilst this description is true of many cases of splenic haemorrhage, it fails to recognize that group of cases in which the clinical course is prolonged. This group is one of considerable importance and the personal experience of 2 cases in a period of three months served as a stimulus to this investigation.

HISTORICAL SURVEY

The picture presented by patients suffering from traumatic rupture of the spleen gained clinical recognition towards the end of the last century. Riegues of Breslau performed the first recorded splenectomy for rupture

in 1893 (Bailey, 1953). In the year 1907 Baudet was able to differentiate clearly between two types of splenic injury. He made a point of the existence of a latent period in the delayed type and, since that time, the delay has often been referred to as the "latent period of Baudet." Baudet's contribution did not recieve great attention, however, as is illustrated by the writings of Moynihan (1911) in Keen's *Surgery* where he discusses diseases of the spleen. Whilst he makes passing mention that splenic haemorrhage may be delayed, he fails to draw any definite distinction between the two groups of cases.

Following that time spasmodic reports of delayed rupture have appeared in the literature. It was, however, not until McIndoe in 1932 collected 45 cases of delayed rupture of the spleen that the condition began to gain general clinical recognition. McIndoe defined criteria requisite for the acceptance of a case as one of delayed rupture and gave an excellent discussion of the pathological features seen in these cases of splenic injury.

In the years that followed the work of McIndoe many reports of single cases and of groups of cases of delayed rupture appeared in various publications. By 1943 Zabinski and Harkins were able to collect no less than 179 well documented reports and were able to add 4 cases of their own. In this paper Zabinski and Harkins give an excellent review of the literature to that date and describe a series of 10 personally treated cases in which there occurred the 4 examples of delayed rupture referred to above. They state that the incidence of a 40 per cent. rate of delayed rupture is the highest reported in the literature and claim that the general rate of incidence is about 15 per cent. The idea that 15 per cent. of cases of splenic trauma show a delayed clinical picture is supported by Ungerstedt of Sweden (1948), but in his paper he only describes one case. In the

present series of cases the incidence was about one-third and is the highest incidence reached in a large group of cases.

It has been stated by Schmid (1935) that there was no authenticated case of delayed rupture of the spleen which has occurred after a period of nineteen days. This is not now true. Attenborough reported 2 cases with latent periods of twenty-two and twenty-six days—in the present series there were 3 cases with latent periods of twenty-one and one case with latent period of thirty-six days (the latter case having been reported in detail by Hueston, 1950) and Olander and Reinmann (1953) report a case operated on two and a half years after injury at which the findings were consistent with long delayed splenic rupture.

Now that the increasing frequency of this type of injury is becoming recognized there seems little point in detailing individual cases and in the present report, the 30 cases of delayed rupture have been reviewed as a group.

treated at the Royal Melbourne Hospital over the period 1930 to 1954 and this group of cases forms the basis for the present investigation.

FIG. II. Photograph of a specimen of delayed rupture of the spleen removed at operation. An intrasplenic haematoma, with subcapsular extension and ultimate rupture may be readily observed. This specimen corresponds to the diagram Fig. III (C).

RESULTS

In this series of cases there were 55 examples of immediate splenic haemorrhage and 30 cases of delayed bleeding, giving an incidence of delayed haemorrhage of approximately 35 per cent. The age distribution in this series is shown graphically in Fig. I. Sixty-seven of these patients were males and 18 females. The age distribution in individual series will clearly vary with the type of work carried out at the institution from which that report originates. The hospital from which this group of cases has been collected deals only with patients over the age of 14 and this fact influences the age distribution to some extent. As can be readily observed the maximum age incidence is in the 20-30 year groups.
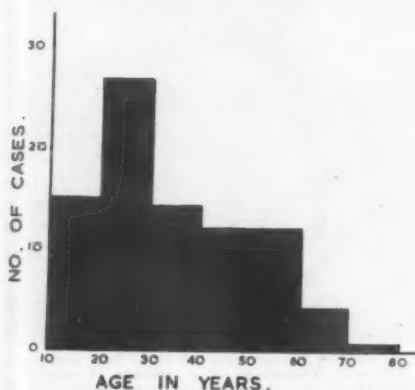

FIG. I. Diagram showing the age distribution of 85 cases of splenic trauma. The peak incidence in the 20-30 age group is quite clear and is made up largely of males.

MATERIAL

Traumatic rupture of the spleen is a relatively uncommon injury and therefore impressions formed by an individual surgeon —based on his personal experience of a few cases—are liable to be fallacious. For this reason more accurate information may be gained by survey of a series of cases as has been done for the present communication. Eighty-five cases of splenic trauma have been

This age and sex distribution is essentially the same as has been observed in other series and probably reflects nothing more than the increased liability of the young male to traumatic episodes. Analysis of the age and sex incidence into 2 groups consisting of those cases showing immediate haemorrhage and those cases showing delayed bleeding shows that age and sex play no part in deciding the nature of the clinical picture.
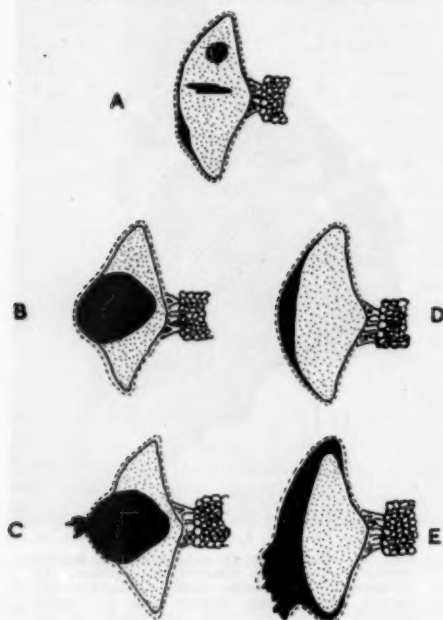
FIG. III. Series of diagrams illustrating the evolution of intrasplenic injuries — the capsule being initially intact. In (A) the diagram shows the spleen immediately after injury. An intrasplenic contusion (in upper pole), an intrasplenic laceration (below upper pole) and a subcapsular haematoma are illustrated. Some of these lesions may retrogress, whereas others will progress. When they do so they follow the pattern illustrated in the further diagrams. (B) shows the developing intrasplenic haematoma having as its origin either an intrasplenic contusion or laceration. In this diagram it has increased in size and is encroaching on the capsule. (C) shows the ultimate rupture of the haematoma with the occurrence of free bleeding. In (D) and (E) are shown the extension of a subcapsular haematoma with stripping of the capsule and ultimate rupture giving rise to free bleeding.

Rupture of the spleen is obviously not confined to adults in its incidence—Scott and Bowman (1946) review the literature on splenic rupture in children and describe 7 cases of their own.

Macroscopic description of lesions found at operation

On examining a series of spleens removed after injury it is found that there is an astonishing degree of variation from case to case. At first sight there may appear to be little or no relation between the various specimens. If, however, the nature of the lesion seen in the spleen be classified as to whether it occurred at the moment of injury (primary lesion) or during the time following the receipt of trauma (secondary lesion), the variety of macroscopic pictures becomes more readily understandable.

FIG. IV. Photograph of a specimen of delayed rupture of the spleen referred to in the text. In this case a subcapsular haematoma had stripped the capsule so extensively that it was found free in the abdominal cavity. The upper specimen is the spleen devoid of capsule and the lower is the capsule with its adherent subcapsular haematoma.

It may be stated at this point that at the moment of impact one or more of 3 primary lesions occur in the spleen. They are:—

(i) lacerations or contusions limited to splenic tissue, the capsule remaining intact;

(ii) lacerations of splenic tissue involving the capsule;

(iii) lacerations of the spleen which in-
volve the hilum and therefore tear
the relatively large vessels at that
site.

Following the initial injury several
secondary phenomena may occur and these
are best discussed under the headings of the
primary injury concerned.

(i) Lacerations or contusions limited to the
splenic tissue, the capsule remaining in-
tact:

In this group there is clearly no free bleed-
ing into the peritoneal cavity from the initial
injury — for this reason there are but few
symptoms and signs to be seen. Indeed, the
existence of the group, as such, would be
somewhat theoretical were it not for the fact
that these intrasplenic lesions may occur in
combination with those seen in groups (ii)
and (iii) and necessitating early splenectomy.

The fate of these cases with an intrasplenic
lesion is variable. Undoubtedly, in some
cases, the intrasplenic or subcapsular effusion
of blood is absorbed completely leaving
merely a scar in the spleen or a collection of
fibrous tissue in the subcapsular region. This
subcapsular fibrous collection accounts for
some cases of "icing sugar" spleen; evidence
as to its traumatic origin may be seen when
histological section is taken, granules of iron-
containing pigment being found scattered
amongst the fibrous tissue. Occasionally cal-
cification in a subcapsular plaque develops.
On rare occasions an intrasplenic haematoma
may undergo incomplete absorption and give
rise to a cyst which persists for some time in
the splenic substance.

On the other hand, many of these cases
progress and lead to secondary rupture with
its associated characteristic clinical features.
The method of progression in these cases is
probably as follows:—

(a) in those cases starting with an intra-
splenic haematoma or laceration the
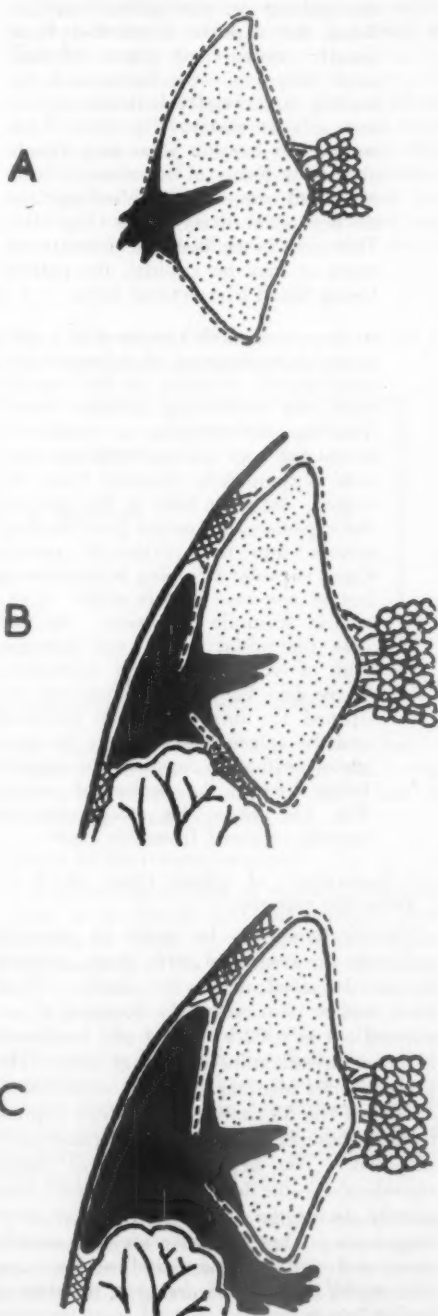haematoma increases in size, gradually

FIG. V. Series of diagrams showing the evolu-
tion of bleeding following a laceration of the
spleen involving its capsule. In (A) a typical
capsular laceration with some degree of capsule
stripping is seen. Whilst free bleeding continues
uninterrupted in many of these cases, it is often
arrested by a combination of clotting and ad-
hesion formation between spleen, diaphragm and
omentum as is shown in (B). At a later time
the encapsulated perisplenic haematoma ruptures,
giving rise to a recrudescence of free bleeding as
is shown in (C).

encroaching on the splenic capsule. When the capsule is reached there usually occurs some degree of capsular stripping — the haematoma extending superficially between capsule and splenic tissue (Fig. II). Ultimately the capsule gives way (probably as a result of interference with its blood supply, and bleeding into the peritoneal cavity occurs (Fig. III). This may be sudden and dramatic in onset or may be gradual, the patient losing blood over several days;

(b) in those cases which begin with a subcapsular haematoma, that haematoma may extend, stripping up the capsule from the underlying splenic tissue. This capsular stripping is variable in extent but may continue until the capsule is completely removed from the spleen. At some time in this process the capsule ruptures and free bleeding occurs into the peritoneal cavity. Gross capsular stripping is uncommon but in one case in this series an extensive example was seen. In this case the initial lesion was pressure with a retractor during operation. Three days later the abdomen was reopened for intra-abdominal bleeding and the spleen was found to be completely without a capsule—the capsule being free in the peritoneal cavity. Fig. IV shows the spleen and its capsule removed from this case.

(ii) Lacerations of splenic tissue which involve the capsule:

Clearly these may be single or multiple and may be associated with those primary lesions described under (i) above. These tears tend to be transversely disposed, a fact pointed out in 1902 by Berger and confirmed by most investigators since that time. The present series also supports this distribution, 32 out of the 55 cases of immediate rupture having fissures which were transversely disposed. The depth of the tear varies considerably—the deepest examples may completely divide the spleen into two or more fragments. These large rents are usually associated with extensive and severe intra-abdominal bleeding occurring at the time of initial injury.
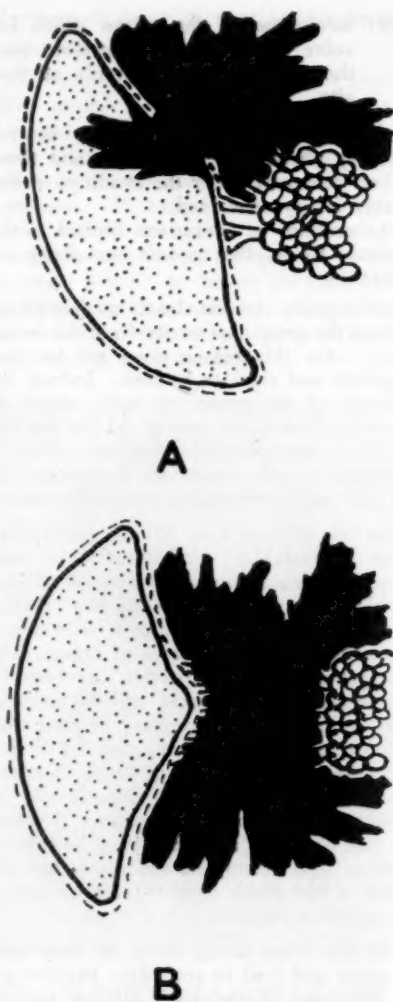

A

B

FIG. VI. Diagrams showing the two types of hilar laceration of the spleen. In (A) a typical incomplete hilar laceration is seen and in (B) a complete avulsion of the spleen with free bleeding is illustrated.

Smaller lacerations may also be of importance. The smallest tear in this series was caused by a biopsy needle inserted into a spleen for diagnostic purposes. Three days later the spleen was removed together with 4 pints of blood from the peritoneal cavity. The sole traumatic lesion to be seen in the spleen at this time was a tiny hole about 1-2 mm. in diameter from which blood was

flowing in a steady stream. Undoubtedly some of these small slits may become temporarily or permanently sealed off and the bleeding controlled. The way in which this occurs is usually by the formation of a clot and/or the formation of adhesions between spleen, omentum, stomach and colon. This method of control of splenic bleeding has been recognized since the time of McIndoe (1932) who gave an accurate description of these events.

The distribution of these various lesions in the 85 cases under review is shown in Table 1.

It is seen that temporary control of bleeding from small lacerations is a much more common cause of delayed haemorrhage than is the rupture of an enlarging intrasplenic haematoma. It is also of great interest that one case with a typical story of delayed rupture, having a latent period of over twenty-

TABLE 1

This table shows the incidence of the various pathological findings as seen at operation in 55 cases of immediate and 30 cases of delayed rupture of the spleen.

Description of Findings	Immediate	Delayed
Single or multiple lacerations	47	—
Single or multiple lacerations plus clotting and/or tamponade	—	18
Hilar laceration	7	—
Hilar laceration plus clotting and/or tamponade	—	4
Splenic avulsion	1	1
Subcapsular haemorrhage with stripping and rupture	—	2
Intrasplenic haematoma followed by rupture	—	5

Whilst in some cases bleeding may be permanently arrested in this fashion, the usual story is that further bleeding occurs after some time, causing breakdown of adhesions with free bleeding into the peritoneal cavity. This progression of events gives rise to a second well-recognized way in which delayed rupture of the spleen may occur (Fig. V).

(iii) Lacerations of the splenic tissue which involve the hilar vessels:

These deserve special segregation into a group because of the fact that laceration of major vessels is an integral part of the lesion. As the most gross example of this type of injury one may find the spleen completely free in the peritoneal cavity — a complete hilar disruption having occurred. There were two such examples in the present series. It is very unlikely that natural arrest of bleeding for any length of time can occur in this group (Fig. VI).

four hours was found at operation to have a complete avulsion of the spleen which was free in the peritoneal cavity. There was no intrasplenic damage in this case.

Nature of the trauma involved

In surveying records of traumatic surgical cases it is found that surprisingly little attention is given to a description of the precise nature of the trauma involved. Often the information given is almost useless. In an attempt to overcome this difficulty, the nature of the trauma involved, in the cases in this series, has been classified into a relatively few groups. These groups have been further divided into two sub-groups, depending on whether an immediate or a delayed haemorrhage was the result of that trauma. The results are shown in Table 2.

On first priciples one might expect that the more severe grades of trauma would give rise to immediate bleeding and the less severe grades to delayed bleeding. Whilst this statment may be true, reference to Table 2

will show that, apart from the fact that in 7 of the cases of delayed haemorrhage trauma was sufficiently insignificant to escape notice, there is little to support the above notion.

remaining cases occurred over forty-eight hours after injury and had a clinical story suggestive of a recrudescence of bleeding from the spleen.

TABLE 2

This table shows the distribution of the various types of precipitating trauma in 85 cases of ruptured spleen.

Nature of Trauma	Total	Immediate	Delayed
Motor accident	28	20	8
Fall from height	30	21	9
Direct blow to left side	16	12	4
Penetrating wound	3	3	—
No known injury	7	—	7
Pressure of retractor at operation	1	—	1

It is certainly true that most severe trauma —sufficient to fracture a pelvis or femur, may be followed by delayed haemorrhage. On the other hand, relatively mild trauma may cause immediate splenic haemorrhage. Quite obviously the question is a complex one and little evidence as to the forces applied to the spleen, as such, can be gained from a description of the forces applied to the body as a whole. The point to be made here is that it is impossible to predict whether a patient will develop splenic bleeding — immediate or delayed — from a description of the nature of his injuries.

CLINICAL PICTURE

As will be shown later the correlation between the clinical story and the pathological findings in these cases of rupture is not always as close as might be expected. A decision must be made, therefore, when dividing cases into the two groups as to what criteria will be adopted in making the division. This is essentially made on clinical grounds and it is therefore reasonable that clinical features should be the deciding factors when subdividing. In this group there were 30 examples of delayed rupture. Two of these occurred in the twenty-four to forty-eight hours period after injury but are included in the delayed series because of the existence of a clear-cut latent period. The

With these points in mind the clinical picture may be discussed under two headings:

(a) Cases of immediate splenic haemorrhage

In all of these cases there had been a story of recent trauma—the delay between injury and surgical treatment was usually in the four to ten hour interval. The distribution of the delay between injury and operation is shown in Table 3.

TABLE 3

Table showing the time-lag from injury to operation in 55 cases of immediate rupture of the spleen. Note that the notion of Berger that 50 per cent. of such cases die within the first hour is shown to be erroneous by this table.

Number of Hours Between Injury and Operation	Number of Cases
0 – 3	8
4 – 5	18
5 – 10	20
10 – 15	3
15 – 20	4
20 – 24	1
During 3rd day	1

In all cases there was some degree of shock. It is to be noted, however, that it was only of sufficient degree to dominate the picture in 15 out of 55 cases of immediate haemorrhage.

The most common complaint made by the patient was that of abdominal pain—occurring in all but 5 of the cases under consideration. The severity and extent of this pain varied considerably — in 34 cases it was experienced under the left costal margin; in 10 it was general abdominal; and in 6 epigastric in distribution.

In addition to abdominal pain patients complained of pain in the left shoulder, or less commonly, in both shoulders — when present this sign is of considerable diagnostic importance.

Examination of the abdomen showed the typical case to case variation in extent of physical signs that is associated with blood in the peritoneal cavity. Tenderness, muscular guarding or rigidity, with some release tenderness, were the commonest findings. In most cases these signs were seen mainly under the left costal margin; in only 15 were the signs gross with general abdominal rigidity. A more important point is that in no less than 10 cases of immediate haemorrhage the clinical findings were quite insignificant. In these cases the combination of shock plus a story of pain under the left costal margin lead to laparotomy at which the diagnosis was made.

(b) Cases of delayed splenic haemorrhage

Turning now to the delayed cases it is seen that the essential difference between these and the immediate cases is to be found in the presence of a latent period. The extent of this latent period shows quite considerable variation as is shown in Table 4.

In 24 of these delayed cases there was a severe recrudescence of symptoms, suggesting a recurrence of severe haemorrhage. In the remaining 6 cases the persistence of vague abdominal symptoms lead to laparotomy during the so-called latent period.

As has been pointed out by other authors the latent period is often not quite symptom free (Hanrahan and Vincent, 1947). In this series there were only 10 cases where the patient had been completely free from pain

during the latent period. The remaining 20 had had some vague upper abdominal pain of minimal severity occurring spasmodically from the time of injury until the time of onset of the severe symptoms. Delannoy (1939) claimed that cases of delayed rupture of the spleen could be divided into two groups on the presence or absence of symptoms during this latent period.

TABLE 4

Table showing the duration of the latent period in 30 cases of delayed rupture of the spleen.

Duration of Delay	Number of Cases
24 – 48 hrs.	2
42 – 72 hrs.	7
During 4th day	1
During 5th day	3
During 6th day	1
During 7th day	2
During 8th day	1
During 10th day	1
During 18th day	1
During 21st day	3
During 31st day	1
No history of trauma obtained	7

Those cases who were absolutely symptom free during the latent period were supposed to show an intrasplenic haematoma which later ruptured through the capsule. Those cases who had minimal symptoms during this interval were stated to have a capsular tear which had ceased bleeding because of clotting and perisplenic adhesions. Whilst this notion is possessed of considerable theoretical appeal, the findings in this series give absolutely no support as to its accuracy — 7 out of the 10 asymptomatic cases showing typical lacerations in the spleen with perisplenic haematoma and adhesions.

At the end of the latent period symptoms return. Shock is not so common as it is in immediate haemorrhage, being present in only 15 out of 30 cases and severe in 2 only.

Pain is a constant clinical finding. In 21 cases it was situated under the left costal margin; in 6 in the epigastrium; and in 2 it was general abdominal. In one interesting case the patient presented with pain and swelling in the left testis, having had a mild abdominal injury some seven days previously. A diagnosis of torsion of the testis was made and the scrotum explored. Blood was found in a hernial sac associated with the testis and was obviously coming from the abdomen. Laparotomy showed a delayed rupture of the spleen to be the cause of his symptoms.

As was seen in cases of immediate haemorrhage, left shoulder-top pain is a common accompanying feature, being present in 21 cases.

Again the extent of the abdominal signs varies from case to case. In only 2 of delayed rupture were the abdominal signs general and extensive. In one case the signs were maximal in the left iliac fossa, and, in another—referred to above—they were concentrated on the left testis. The remainder showed tenderness, muscular guarding or rigidity and release tenderness localized to the area under the left costal margin. In 3 of these cases was a palpable subcostal mass detected.

SPECIAL TESTS

During the latent period, serial estimation of haemoglobin level may show progressive diminution and enable surgical intervention to be performed before a severe recurrence of bleeding occurs. This test has been mentioned by several authors but was not used in any case in the present series. Although no use was made of radiography in diagnoses in the present series, it would appear that serial radiological investigation of the left subphrenic area may be of value during the latent period.

Thomas, Reinhart and Gershon-Cohen (1952) have described a characteristic triad of findings in which there is progressive elevation of the left hemidiaphragm, depression of the gas bubble in the stomach and of the gas in the splenic flexure as seen on serial X-rays. They state that these findings, in the presence of a history of trauma to the region of the left flank, warrant laparotomy.

Peritoneal tapping, as recommended by Byrne (1950), may be of value in some cases but would appear to be dangerous as a routine diagnostic measure.
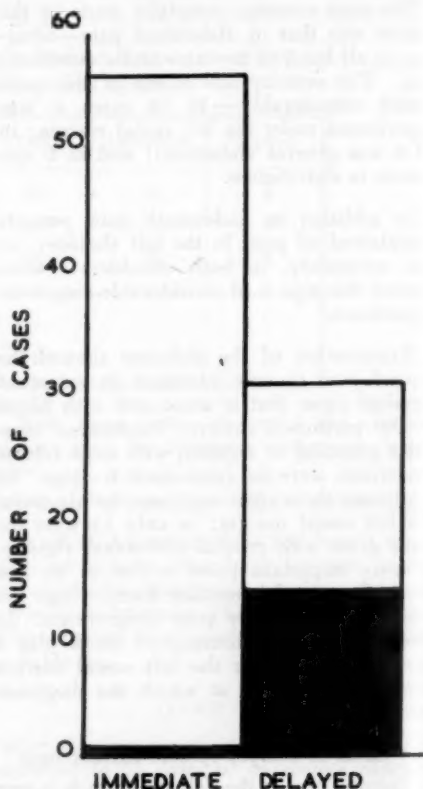

FIG. VII. Diagram showing the relative proportion of immediate to delayed cases of splenic haemorrhage. On the same graph the cases which were incorrectly diagnosed are shown in black. The increased liability to misdiagnosis in cases of delayed haemorrhage is obvious.

O'Connell (1946) has described a sign—that of the occurence of left shoulder tip pain when the patient is placed in a head-down position which, he states, indicates the presence of fluid in the peritoneal cavity. No use has been made of this sign in the present series.

That the frequency of delayed splenic haemorrhage is not appreciated is well illustrated when the pre-operative diagnosis is examined in these cases of ruptured spleen.

Whereas an incorrect pre-operative diagnosis was seen only in one out of 55 cases of immediate haemorrhage, there were no less than 13 misdiagnosed cases of delayed splenic haemorrhage in a series of 30 (Fig. VII). The most common diagnostic error was that the delayed case was mistaken for perforated peptic ulcer.

TREATMENT

Quite clearly a major part of the management of splenic bleeding is the resuscitation of the patient. In cases where four or more pints of blood have been lost into the peritoneal cavity this must be replaced if satisfactory recovery is to occur. The incision used depends largely on the accuracy of diagnosis. A left subcostal approach gives excellent access to the spleen but is not really satisfactory if lesions elsewhere require attention. A left paramedian incision does not have this defect even although it gives less adequate exposure of the splenic pedicle. The left subcostal muscle-splitting incision advocated by Larghero Ybarz and Giuria (1951) should give adequate access but it is difficult to agree with these authors when they state that should a splenic rupture be diagnosed via a paramedian incision, then that incision should be closed and a muscle-splitting one made.

Much blood is usually found in the abdominal cavity at laparotomy. In the delayed cases some of this blood is recent whilst in other areas it has obviously been there for some time.

Careful palpation of the surfaces of the spleen in order to recognize tears in its substance is widely practised and its value has been emphasized by several authors. The manoeuvre of rupturing the lienorenal ligament in order to deliver the spleen rapidly through a paramedian incision is also too well recognized to need further description.

It is generally agreed that splenectomy is the best method of treatment of this lesion and it has been the only method of treatment employed in the present series.

In one interesting case (not included in the figures because of the doubt about diagnosis) a palpable mass developed in the left upper quadrant following trauma to that

area. Owing to the good general condition of the patient, laparotomy was deferred only to find that the mass gradually disappeared without further episode suggestive of recent bleeding. The surgeon in charge of this case suggested that it was one of splenic haemorrhage.
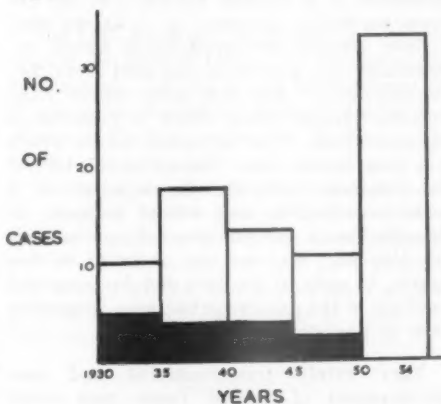

FIG. VIII. Diagram showing the incidence of 85 cases of splenic rupture in five year periods as seen at the Royal Melbourne Hospital since 1930. On the same graph the mortality rate is illustrated in black. The steady diminution in mortality from this disease is obvious and is almost certainly due to modern methods of resuscitation coupled with advances in anaesthesia.

MORTALITY

In this series of cases there were 16 deaths —a mortality of approximately 20 per cent. As can be seen in the graph (Fig. VIII) this mortality has steadily declined with the passage of time and, in the last three and a half years, there have been 33 cases of splenic haemorrhage treated without death. The main cause of death in this condition is blood loss and undoubtedly modern methods of resuscitation, coupled with newer methods of anaesthesia, are mainly responsible for the reduction in mortality. The reason for death in the 16 cases was as follows:—

13 died as the result of blood loss;

2 died from severe chest infection (in the pre-antibiotic era);

1 died from paralytic ileus.

It is to be noted that 4 of the deaths occurred after delayed haemorrhage, thus emphasizing the need for early diagnosis and treatment of this condition. Post-operatively

the most common complication is that of infection at the base of the lower chest. This was described as a clinical complication in 15 cases in the present series.

In the remote post-operative period, the occurrence of intestinal obstruction due to adhesions is a definite hazard. In the 69 cases surviving operation it is known that 5 have already developed small bowel obstruction (one patient having died from this complication). The only other remote post-operative complication which is recorded is the occurrence of an incisional hernia which was seen in one case. Injury to the tail of the pancreas, with the development of a pancreatic fistula and wound rupture, is described as a complication of splenectomy for trauma. No case was observed in this group, in spite of the fact that bruising and swelling of the pancreatic tail were frequently seen at operation.

Very rarely transplantation and new development of splenic tissue may occur following rupture — the literature on this interesting complication has been recently reviewed by Wise (1953).

DISCUSSION

McIndoe in 1932 laid down three criteria for the acceptance of a case as one of delayed rupture of the spleen. These are:—

(a) that the case must have been of over forty-eight hours' duration;

(b) that the history must indicate that a secondary haemorrhage has occurred;

(c) that all cases where the haemoglobin gradually sank over a period of days, indicating a slow, continued bleeding, be excluded from the series.

McIndoe also describes both the rupturing of an intrasplenic haematoma and the cessation of bleeding with a perisplenic haematoma as the operative findings in this group of cases and infers that the two have close association. However, the relationship between the morphological findings at operation and the clinical story are not always as close as might be supposed and it would appear reasonable to discuss at some length the criteria for the description of a case as one of delayed haemorrhage.

In choosing the time interval of forty-eight hours McIndoe admits that an arbitrary decision is involved. There is no reason to suggest why an intrasplenic haematoma may not rupture within the time interval of forty-eight hours, and — provided that a definite clinical latent period exists — there is no reason why this case should not be included under the heading of delayed rupture. But, by admitting cases under the forty-eight hour period occasional examples are seen where the findings at operation are surprising. Jenkins (1953) reported a case of twenty-four hours' duration with typical latent period in which, at operation, the lower lobe of the spleen was found free in the peritoneal cavity. In the present series, a case of twenty-four hours' duration with typical latent period, was found to have a complete hilar tear at operation. The mechanism of delay in these cases is probably that traumatic arterial spasm delays the onset of severe bleeding.

It has been stated already that the findings in most cases of delayed rupture are those of vessel spasm, splenic laceration, perisplenic haematoma, clotting and adhesion formation, with the later recrudescence of haemorrhage giving rise to the typical flare up of symptoms. Apart from the fact that the vessels involved in these cases are smaller and that the delay is longer than in the 2 cases referred to above, there is no essential difference in the sequence of events taking place. For these reasons it is submitted that a case should be accepted as one of delayed rupture in the time interval under 48 hours, provided that there is a definite clinical history of a latent interval (if a 48-hour limit is accepted as minimal, only 2 cases would be excluded from the present group).

Let us turn now to the second criterion: the clinical story must indicate the occurrence of a secondary haemorrhage. Whilst this criterion is essential in those cases which occur under the two to three day limit after injury, it is not necessarily so in cases which remain unoperated on after that time. Most authors agree that the interval of Baudet is usually not quite asymptomatic. During that period of time reactionary oedema and swelling of the spleen will occur, associated perhaps with a small amount of continued

bleeding. Should such a case be left then, ultimately a catastrophic bleeding with a severe recrudescence of symptoms will occur.

With the present modern diagnostic methods available it is quite possible that the alert clinician will be able by the use of serial radiology, to diagnose a ruptured spleen during this latent interval. The mere fact that operation be carried out before the onset of severe symptoms should surely not exclude such a case from a diagnosis of delayed rupture.

With the final criterion that no case showing progressive and steady deterioration since the injury should be included as one of delayed injury there is little argument.

The next problem for discussion is that of the occurrence of splenic haemorrhage without any obvious history of trauma. These have been regarded by some as examples of spontaneous rupture of the normal spleen—it is, however, highly improbable that such an entity exists. Numerous reports are found in the literature where a post-operative history of trauma is obtained, that history having been overlooked by the patient pre-operatively because he regarded it as being of no importance.

Further, if any time has elapsed between the occurrence of trauma and the onset of bleeding, the injury is often forgotten altogether. Baillie (1952), in reporting a case where the closest of questioning was required in order to elicit a story of trauma, remarks that careful enough questioning will always reveal a story of injury in all cases of supposed spontaneous rupture of the normal spleen. If one pauses to consider the frequent occurrence of minimal trauma to the trunk during daily life, it must be admitted that to completely exclude such an episode from a case of splenic bleeding would be virtually impossible.

To develop this argument further, it is almost certain that all these cases of supposed spontaneous rupture of the normal spleen are, in fact, examples of delayed traumatic rupture of that organ. Clearly if symptoms occur immediately on the receipt of a traumatic episode, the patient will connect the trauma with his symptoms. The fact that no traumatic episode is remembered is good evidence that a typical latent period has occurred. Moreover, the findings at laparotomy in the 7 cases seen in this series would indicate that bleeding had occurred over a period of some days.

The high incidence of the delayed clinical picture in splenic trauma is not generally appreciated. In this paper an incidence of 21 examples of classical delayed rupture in a group of 85 cases is found. If the 2 cases which occurred under 48 hours and the 7 where no traumatic episode was detected be admitted to the figures (as indeed they should be) the incidence rises to 30 cases in the 85. Thus about one case in 3 presents the delayed picture and appreciation of this point is important, particularly to those who have the duty of teaching students.

Summary

1. A series of 85 cases of traumatic rupture of the spleen is reviewed.

2. It is emphasized that the clinical picture of this injury is often not as clear cut and dramatic as is generally supposed. (In only 19 cases in this series was the shocked condition of the patient of sufficient severity to dominate the picture and often the physical signs to be found in the abdomen were minimal.)

3. The diagnostic value of pain in the left shoulder, when present, is stressed.

4. The high rate of occurrence of a delayed clinical picture (approximately one case in 3) is emphasized.

5. The various morphological pathways of evolution of splenic rupture are discussed.

6. It is emphasized that the division of cases of splenic rupture into the immediate and delayed varieties is essentially one based on clinical features and the factors influencing this decision are described.

7. A brief review of the literature, placing particular reference on that pertaining to delayed rupture, is given.

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VOLVULUS OF THE CAECUM

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VOLVULUS of the caecum (Footnote *) is an uncommon condition, its incidence being less than one per centum in any large series of acute intestinal obstruction. Three hundred odd cases only have been reported in the surgical literature (Dean, 1952) so that it is not out of place to discuss five further cases of this interesting cause of acute intestinal obstruction.

CASE REPORTS
(Footnote †)

Case 1

Volvulus of the terminal ileum, caecum, ascending colon and right half of the transverse colon.

The patient was a male, aged 62 years, who had always been well until this illness.

Four days prior to his admission to hospital, he first experienced a constant, aching pain round the umbilicus. He was also constipated, a most unusual condition for him. He then took some castor oil which had no effect save to accentuate the pain. After two days, the pain spread all over the abdomen. It became very severe and was now intermittent and colicky, and associated with progressive abdominal distension. Though still constipated, the patient now passed a little mucus per rectum very few hours. On the third day of his illness, he vomited once for the first and only time.

Examination on admission on the fifth day of his illness, disclosed an ill, dehydrated man whose

Footnote * — The term "volvulus of the caecum" includes, firstly, torsion of the isolated caecum; secondly, that of the caecum plus a variable amount of the terminal ileum and ascending colon; and thirdly, the latter combination plus the right side of the transverse colon.

Footnote † — Cases 1 and 2 were under my care, the first at the Royal North Shore Hospital, the second at the East Ham Memorial Hospital, London. This latter patient was reported previously (Rose, 1941) as an example of retroposition of the transverse colon complicated by caecal volvulus so that a summary only is presented here. Cases 3, 4 and 5 were abstracted from the records of the Royal North Shore Hospital, being, with Case 1, the only examples of this condition seen there in the past ten years.

tongue was furred and dry. His pulse rate was 100 per minute, his respiratory rate numbered 30 per minute, and his temperature was normal.

The relevant physical findings were confined to the abdomen and chest. There was generalized abdominal distension present, most marked in the epigastrium and left upper abdominal quadrant. Generalized tenderness was elicited but there was no muscle rigidity. The percussion note was tympanitic, and increased borborygmi were heard on auscultation. A rectal examination showed no abnormality save for the presence of some mucus in the rectum.

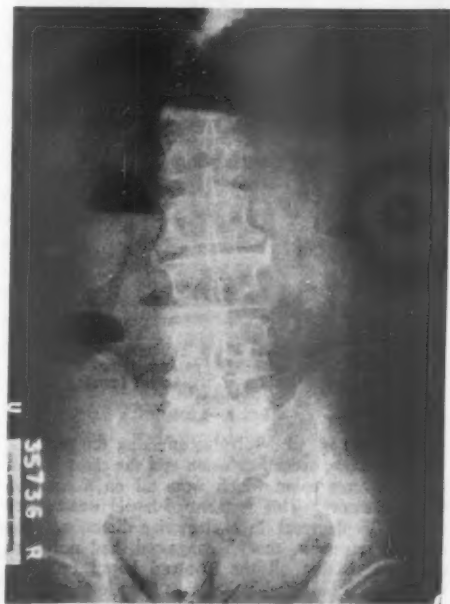Examination of the chest showed some dulness to percussion and diminished air entry at the left base posteriorly.

FIG. I. Case 1: Plain radiograph of the abdomen taken with the patient in the erect position showing the large fluid level of the caecum in the left hypochondrium together with fluid levels in the small bowel.

A plain radiograph of the abdomen revealed a large gas containing loop of bowel with a fluid level in the left hypochondrium. There were smaller fluid

levels elsewhere in the abdomen. The small bowel was dilated. (See Fig. I, taken with the patient in the erect position, and Fig. II, with the patient supine.)

A stomach tube was passed and only a few millilitres of fluid were obtained so that it was obvious the bowel involved was not the stomach.

As soon as intravenous water and electrolytic therapy was established, the abdomen was explored through a right paramedian incision.
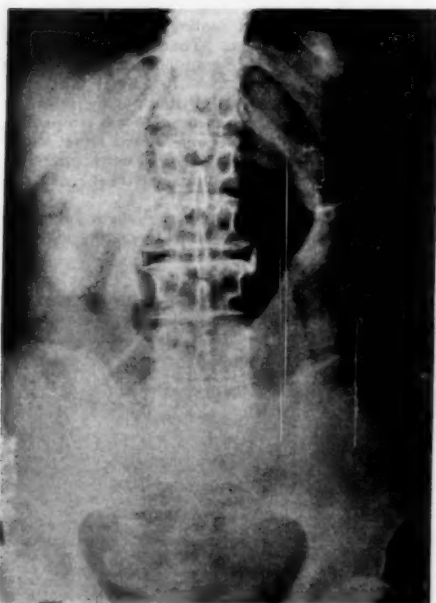

FIG. II. Case 1: Plain radiograph of the abdomen taken with the patient lying supine showing the distention of the bowel forming the volvulus.

There was found a volvulus affecting the terminal ileum, caecum, ascending colon and the right half of the transverse colon which were all on a common long mesentery. This segment of bowel was twisted twice (720 degrees) in a clockwise direction. The mesenteric vessels, though congested, had not been occluded so that all the involved bowel was still viable. The bowel with the large fluid level was the caecum situated high up in the left hypochondrium. It was so over-distended with fluid and gas that it was pressing on the diaphragm, so explaining the diminished air-entry into the lower lobe of the left lung.

The caecum was decompressed by aspiration as its walls were paper thin and seemed on the point of imminent rupture. The volvulus was then untwisted and the caecum was placed in the right iliac

fossa. A tube caecostomy was performed using a stab incision in the right iliac fossa for the passage of the tube.

Convalescence was uneventful save for a mild *Escherichia coli* wound infection. The lung expanded within two days and the respiratory rate dropped to 20 per minute. The caecostomy closed spontaneously in sixteen days.

A follow-up eighteen months later disclosed that the patient had had no further abdominal trouble.

Case 2

Volvulus of the terminal ileum, caecum, ascending colon and right half of the transverse colon complicating retroposition of the transverse colon.

The patient, a previously healthy man, aged 43 years, had a three day old history of severe abdominal pain commencing in the left iliac fossa and radiating all over the lower abdomen. It was colicky in nature and recurred in spasms. With each attack of pain, the abdomen became distended, the distension subsiding as the pain eased. These attacks were becoming more violent and prolonged. He had been constipated for the previous two days.

Examination disclosed that the abdomen was distended, especially below and to the right of the umbilicus. This area was tender and it felt tense; the tenseness relaxed between the spasms of pain which were recurring every few minutes. The percussion note was tympanitic and borborygmi were present.

At operation, the terminal ileum, caecum, ascending colon and a portion of the right half of the transverse colon were found to form a volvulus which had twisted 360 degrees clockwise on a transverse axis so that the caecum was to the right of the umbilicus. The bowel concerned, especially the caecum, was of paper thinness due to the enormous distension, but it was still viable. Distal to the volvulus, the transverse colon was found to travel behind the superior mesenteric vessels to disappear through a tunnel in the small bowel mesentery.

The volvulus was untwisted after aspiration of the caecum, which was then returned to its normal position. Tube caecostomy was then performed.

Convalescence was uneventful. It was impossible to do a follow-up owing to the onset of World War 2.

Case 3

Volvulus of the terminal ileum, caecum, ascending colon and right half of the transverse colon.

The patient was a male, aged 50 years, who had intermittent generalized, colicky abdominal pains lasting half an hour, at irregular intervals for one week. The abdomen became distended with each

pain and as the pain subsided so did the distension. Ten hours prior to entering hospital, the pain became continuous and severe. It was associated with vomiting and increasing distension. The pain was now localized to the lower abdomen, whereas the tenderness and distension were generalized.

Operation revealed a condition similar to that found in Case 1. The volvulus was untwisted and a caecostomy performed with a satisfactory result.

Case 4

Recurrent volvulus of the terminal ileum, caecum and ascending colon.

Ten years previously, this 70-year-old man had had a volvulus of the terminal ileum, caecum and ascending colon. This was treated by untwisting the volvulus, but neither caecostomy nor any other method of caecal fixation was performed.

He was then well until four days prior to this admission, when he commenced to suffer attacks of severe, generalized, colicky abdominal pain accompanied by ever-increasing abdominal distension and vomiting.

On admission, he was seen to be very ill with a grossly distended abdomen. There was generalized abdominal tenderness and rigidity.

Operation revealed a general peritonitis due to small bowel contents lying free in the abdominal cavity. They had escaped through a perforation in the centre of a small area of gangrene in the caecum. This organ was present in the right hypochondrium and was involved in a volvulus consisting of the terminal ileum, caecum and ascending colon. This had twisted 360 degrees in a transverse axis.

The volvulus was untwisted, the area of caecal gangrene excised and a caecostomy performed through the deficiency in the caecal wall.

However, the patient died shortly afterwards from the effects of the general peritonitis.

Autopsy revealed no cause for the volvulus other than the presence of the primitive mesentery of the involved bowel.

Case 5

Volvulus of the terminal ileum, caecum and ascending colon.

This patient, a male aged 63 years, for nine months prior to admission, had a history of intermittent, generalized, colicky abdominal pain lasting several hours at intervals of days to weeks. Each attack was associated with distension, constipation and the occasional passage of mucus per rectum. As the pain and distension subsided, there would occur profuse diarrhoea with liquid motions.

His present attack had lasted five days and on admission he was seen to be suffering from a generalized peritonitis with an enormously distended abdomen.

Abdominal exploration disclosed that there was a volvulus of the terminal ileum, caecum and ascending colon with the caecum in the left upper quadrant. A portion of the anterior wall of the caecum was gangrenous and in the middle of this was a perforation through which had escaped much faecal content resulting in general peritonitis.

The volvulus was untwisted and the gangrenous caecal area excised. The resultant deficiency in the caecal wall was used to perform a tube caecostomy.

However, the patient died a few hours after operation.

Autopsy revealed no cause for the volvulus save for the long ileo-colic primitive mesentery.

DISCUSSION

Aetiology

Volvulus of the caecum can only occur when its primitive mesentery persists. It is obvious, however, that this is only a predisposing cause, because this embryological structure may be found to persist in as much as 10 to 30 per cent. of autopsy material whereas volvulus of the caecum is quite uncommon. In most cases, however, no exciting cause can be found. Desporges and Wilson (1953) describe a patient the exciting cause of whose volvulus the authors thought to be an obstructive carcinoma of the descending colon. In Case 2 of this article, the exciting cause may have been the retroposition of the transverse colon which caused a persistent spasm of the transverse colon as shown by later opaque enema.

In some cases, pregnancy acts as an exciting cause (Sheldon, 1944) as it may in volvulus of the small bowel (Bellingham, Mackey and Winston, 1949).

It would be of interest to know why volvulus of the caecum does not occur until an adult or even old age is reached, when the acknowledged predisposing cause, the persistence of the primitive mesentery must have been present all the patient's existence. Possible the mesentery lengthens as age increases. On the other hand, so-called idiopathic volvulus of the small bowel, where the mesentery is normal, so that there is neither predisposing nor apparent exciting cause, may occur at any age from infancy onwards.

Mechanics

The actual length of gut involved in the twist of the volvulus depends on the length which is on a free mesentery. It may be the caecum only, or the terminal ileum, caecum and ascending colon (Cases 4 and 5), or the terminal ileum, caecum, ascending colon and the right half of the transverse colon (Cases 1, 2 and 3).

The type of volvulus is determined by the axis of rotation. Graham (1930) pointed out that there are three types:

(1) One whose axis of rotation is transverse and the caecum rotates so that its posterior surface looks forwards (that is, if it only rotates 180 degrees). In these cases, the caecum is found on the right side of the abdomen (Cases 2 and 4).

(2) The axis of rotation is oblique and the caecum ascends towards the left hypochondrium with its posterior surface anterior and its lower pole uppermost.

(3) Rotation occurs about the long axis of the caecum, usually clockwise.

In Cases 1, 3 and 5, the mechanism appeared to be a combination of (2) followed by (1). In Case 1, the position of the caecum and its distension caused pressure on the left lung base through the diaphragm.

The first 180 degrees twist of the volvulus obstructs the ascending colon but not the ileum whose contents pass on and cause the enormous distension of the caecum. The second 180 degrees twist obstructs the ileum and a closed loop obstruction occurs which then becomes strangulated as the twist tightens the mesentery and occludes the vessels. It can then be seen that the longer the length of bowel implicated, the longer the clinical course may be before strangulation and hence gangrene and rupture of the volvulus occurs. A slow twist may have a viable bowel even after three or four days (Cases 1 and 2). This differs from volvulus of the small bowel where viability is a matter of hours and not days.

Age

The ages of the cases presented here were 62, 43, 50, 70 and 63 years respectively, whereas most authors state that it occurs in young adults (Corner and Sargent, 1905; Dean, 1952).

Symptoms and physical findings

The symptoms depend on the speed of formation of the volvulus and the occlusion of the mesenteric vessels. A slowly forming volvulus causes mild symptoms initially, whereas a quickly forming one causes severe symptoms from the start.

As in all cases of mechanical intestinal obstruction, pain is the first symptom. As the volvulus forms, it causes obstruction so that there will be intermittent colicky pain due to obstructive peristalsis of the bowel proximal to it. As the mesentery is tightened there will be acute severe continuous abdominal pain due to this and the pain may radiate through to the back as the root of the mesentery is involved. In addition, there is the continuous pain of the tense overdistended caecum. If this ruptures, the pain of general peritonitis supervenes while the pain due to the now deflated caecum lessens. Consequently, each case will differ in the pain suffered according to the combination of factors present.

Constipation is present with the obstruction. However, in Case 5 profuse diarrhoea followed the subsidence of the pain and distension obviously due to the caecum discharging its contents into the colon as the volvulus spontaneously untwisted itself. In Case 1, mucus was passed per rectum during the obstruction.

Some patients have a history of frequent attacks of partial volvulus which subside spontaneously until the one which brings them to operation (Cases 3 and 5). These attacks are characterized by generalized colicky abdominal pain and distension which subsides as the pain lessens.

The onset of the main attack, in which the volvulus fails this time to untwist itself, is often slow, as in Case 1, where two days

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aching pain occurred before the severe colic commenced, or the pains are severe and colicky from the start. The pains may be generalized from the commencement (Cases 1, 3, 4 and 5), or start in the umbilical region or even in the left iliac fossa (Case 2).

Once the pains become severe as the volvulus tightens, they are associated with marked and progressive abdominal distension. This is due, firstly, to the enormous distension which the caecum suffers early, and secondly, to the obstructive distension of the small bowel, a later sign. Distension is thus first localized to the position of the caecum, and then it becomes generalized as the small bowel dilates. The dilated caecum may even feel like a cystic tumour or a tense balloon depending on its tension. · Whilst the distension is still localized to the caecum, one may hear the exaggerated borborygmi from the obstructed but as yet undilated small bowel.

Vomiting is an inconspicuous feature of caecal volvulus. There is little early reflex vomiting as seen in acute volvulus of the small bowel. There may be vomiting due to the pressure of the distended caecum on the stomach when it is displaced to the left hypochondrium. There is later vomiting due to the intestinal obstruction.

Tenderness is an early sign as in all cases of threatened bowel strangulation. It is found over the volvulus and becomes more generalized as more bowel is involved. It becomes more marked as the volvulus becomes tighter.

Rigidity is a late sign betokening rupture and peritonitis (Cases 4 and 5).

A radiograph of the abdomen will clinch the diagnosis of caecal volvulus by showing the long fluid level of the dilated caecum. Should this be in the left upper abdominal quadrant (and according to McGraw, Kremen and Rigler [1948], it is found there in 90 per cent. of these cases), at first sight it may appear to be stomach, but aspiration of this organ will rapidly solve that problem (Case 1).

Treatment

Immediate laparotomy is necessary to untwist the volvulus. The caecum may have to be aspirated before this is done to decompress it and render it less likely to burst.

As the bowel may remain paralyzed and distended for some time afterwards, and as stomach or small intestine suction will not drain the caecum or large bowel, a tube caecostomy should be performed to keep the caecum empty. This will also help to anchor this organ by the formation of adhesions between it and the anterior abdominal wall and so prevent a recurrence. This was not done in Case 4 and a recurrence did indeed take place.

Should the bowel be non-viable, then resection must be done. One may be able to limit this to the gangrenous area of the caecum alone and use the resultant opening for a caecostomy tube.

Mortality

The reported mortality of this disease is high, and in this series, two of five died (Cases 4 and 5). Peritonitis is the usual cause and is due to the rupture of the gangrenous bowel.

Death is not due to anything peculiar to caecal volvulus *per se* but to the lateness most of them reach hospital, for instance, the cases reported here were not seen in hospital until 96, 72, 168, 96 and 120 hours respectively had elapsed from the commencement of their illness, that is, all these cases were sent in too late. This, I feel sure, is due to two factors, firstly, the often slow onset of this condition and the early abdominal distension lull the referring practitioner into believing he is dealing with a large bowel obstruction, at this age probably due to carcinoma; secondly, for some unknown reason, many practitioners believe it is safe to temporize with large bowel obstruction and so miss the urgent nature of the case.

Thus earlier diagnosis will bring the mortality down to reasonable figures so that cases such as Case 4 and 5 will no longer occur.

SUMMARY

Five cases of caecal volvulus, one associated with retroposition of the transverse colon, are discussed.

This condition, to date, has a high mortality due, not to anything peculiar to caecal volvulus *per se*, but to delay in its diagnosis.

ACKNOWLEDGEMENTS

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METAPLASIA IN THE TUNICA VAGINALIS TESTIS

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THERE has been a gradual but general change, in the last few years, from the rigid views of the early part of the century regarding the possibility of changes in tissues of the body in adult life. Although it is still stated or implied in some papers that any tissue not normally found in an organ or area must have developed during pre-natal life, there is an obviously increasing body of opinion in the direction of assigning this formation, in many cases, to local post-natal development.

That changes in tissues, previously regarded as impossible, do occur have come to be accepted largely because direct observations have been made much more frequently. Review of the literature shows that occasional cases of aberrant tissue (commonly occurring more frequently in old people than in the young) were observed in the last century; these received but little attention. However, with the greater number of investigators and the, in some respects unfortunate, larger amount of published material in recent years examples of metaplasia can no longer be ignored.

The development of stratified, and sometimes squamous epithelium has been one of the frequently observed phenomena and therefore has been widely accepted and with greater readiness than has been the case with other changes. Thus it has become appreciated that the occurrence of stratified squamous epithelium in the superficial tissues does not imply that squamous epithelium is necessarily always of ectodermal origin. In normal circumstances it occurs in structures known to be endodermal. Despite this, curious deductions are still sometimes drawn from the presence of stratified squamous epithelium.

In pathological conditions its development in the alimentary canal is well known and is now usually accepted without question. Its presence, in certain circumstances, in the renal tract and the uterine cavity is discussed in every textbook dealing with these subjects so that stratified epithelium, which often is even of a squamous type, is recognized as being derived easily from mesodermal structures.

Even better examples of such metaplasia (better in that they involve greater change) have been described in the serous cavities—a phenomenon noteworthy not only because of the considerable morphological difference between the serosal cells and stratified epithelium but also because any of the stimuli (usually of a mechanical type) associated with the development of stratification are ordinarily less likely to be effective here. Such stratification in the peritoneum has been demonstrated by Crome (1950).

Cases of development of stratification going on the squamous metaplasia in the lining of hydroceles of the spermatic cord have been described earlier (King, 1951). The degree of change is different in different cases but the actual differences, providing gradations from the slightly hyperplastic serosa up to well-developed stratified squamous epithelium, themselves indicate the manner in which the change probably occurs.

From the observation of the cases mentioned it is apparent that there might be, in appropriate circumstances, a similar development in the lining of the tunica vaginalis testis. The close relation of the two developmentally (and their similarity in structure) makes the occurrence almost certain; however it is essential that the matter should be one of demonstration in every place and this is all the more important when the condition is observed only infrequently.

The activity of any tissue depends on its environment and this, of course, is in normal circumstances such as to maintain the "normal" appearances. This is what has given rise to the view in the past that tissues are capable only of limited activity. It is unknown to what extent this activity is limited but is clear that the limitation is much less

FIG. I. Photomicrograph of portion of the wall showing (on the right side) serosa which merges into a double- and then multi-layered epithelium. (x 25)

than was previously imagined. The observation of pathological conditions demonstrates that the changes encountered are indeed possible and this is why the description of the uncommon and peculiar pathological change is so important.

An example of this squamous metaplasia in the lining of the tunica vaginalis is described here.

FIG. II. Photomicrograph of part of the wall showing a stratified epithelium. (x 25)

CASE HISTORY

The patient, aged 49, had had an enlargement of the right side of the scrotum for several (about four) years. The precise time of onset and mode of development was uncertain but a swelling was noticed, without any pain, and this remained the same size for the whole period until, over a period of three months, it began to become slightly further enlarged.
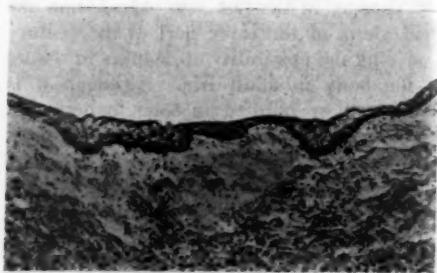

FIG. III. Photomicrograph of part of the wall showing an epithelium with a well defined basal layer; the number of layers of cells varies in different parts. (x 25)

When examined there was moderate enlargement of the right side of the scrotum and a poorly translucent swelling, in front of and slightly above the testis, was found. Operation was advised and removal of most of the wall of the cyst lying anterior to and in contact with the testis was carried out.
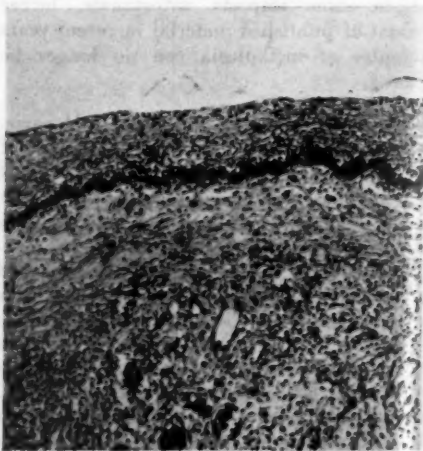

FIG. IV. Photomicrograph of an area of the wall where the epithelial lining is thick. There is a considerable resemblance to some mucous membranes. (x 25)

The specimen, after operative removal, consisted of thickened tissue apparently of a cyst wall with a considerable defect in one portion. The wall was thickened and fibrous and most of the lining was smooth but in some parts laminated superficial material could be peeled off.

Histologically, the tissue was largely fibrous in character but in one part there was portion of epididymis which showed inflammatory changes of a non-specific type.
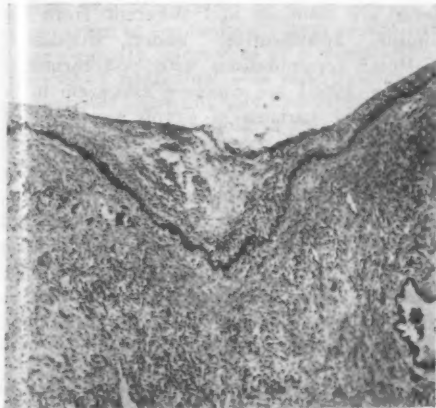

FIG. V. Photomicrograph of an area showing irregular and gross thickening of the epithelial layer. (x 25)

On the internal aspect of the tissue there was a definite lining. In a considerable part, the cells were of the typical serosal type but in many areas they comprised at least a double layer (Fig. I). In other parts there was an increase in the number of cells and these were stratified (Fig. II). Sometimes there was a sharp demarcation of the stratified epithelium from the flattened serosal cells (Fig. VI). These were most prominent in the region adjacent to the epididymis.
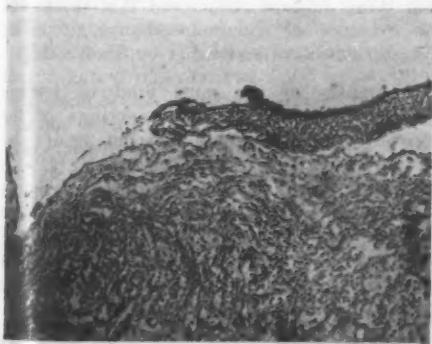
The number of layers varied irregularly in the different parts and in some areas the deepest layer formed an undulating border (Figs. III and V), though definite papillae were not present.
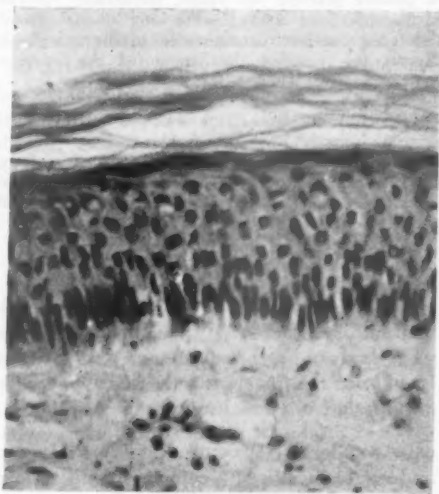

FIG. VII. High power photomicrograph of a part of the wall which showed more definite prickle cells and keratin formation on the surface. (x 380)

The cells, in general, were polyhedral though the superficial ones were flattened. The appearance was that of the epithelium of the vagina or other mucous membranes (Figs. IV and V).

Although in many parts the cells were merely stratified, in other places there were well-defined regular intercellular prickles, eleidin granules and lamellae of homogeneous material similar to that seen on the skin or other squamous epithelia (Fig. VII).
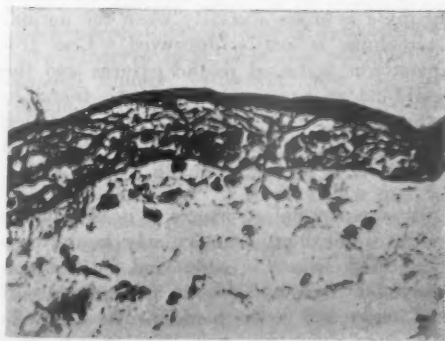

FIG. VI. Photomicrograph of portion of the wall showing an abrupt transition from the serosa to stratified epithelium. (x 50)

FIG. VIII. Photomicrograph of a section of the wall stained by the ferricyanide method of Chèvremont and Fréderic (the tissue is otherwise unstained). There is pigmentation of the superficial layers of the epithelium. (x 350)

Special stains for keratin were used. These, for the most part, were those such as the ferricyanide method of Chèvremont and Fréderic (1943) which is a specific histochemical test for sulphydryl radicals. A positive reaction was found, remarkably specific to the superficial layers, in the intercellular cement substance but best demonstrated in the superficial layers; this reaction thus supported the morphological evidence (Fig. VIII).

The layer of epithelium was, in some places, separated partly from the deeper tissues — demonstrating a clear distinction of the epithelium from this adjacent connective tissue (Fig. VIII) and, in some areas, where the epithelium had disappeared there was no covering to the connective tissue.

DISCUSSION

Stratification of the simpler forms of epithelium is a universal phenomenon in the adult body. Furthermore, not only does it occur on the external coverings but also develops in internal linings.

In ectodermal structures it is well known and has always been accepted without serious question. Thus it develops in sebaceous glands and sudoriferous glands and, though less common, is well known in the breast. It is a relatively common phenomenon in the salivary glands. In all of these it is frequently squamous in form and well-defined keratin is usually demonstrable.

In structures of endodermal origin it is well known in the thyroid gland and indeed here can be caused to develop under experimental conditions. It occurs commonly in the ducts of the pancreas and in the bronchi. In these it arises specially when the normal epithelium is partly destroyed. Less frequently it is found in the pylorus and the gall-bladder and, indeed, may be found in other parts of the alimentary canal. Often this epithelium is squamous in form and keratin is to be found.

In mesodermal structures it occurs in the body of the uterus particularly in the condition of *kraurosis uteri* and in the epididymis. It is well known in the pelvis of the ureter and in the bladder. As stated, in all these cases not only does stratification occur but a special differentiation with formation of keratin is a common and generally accepted phenomenon.

The position is somewhat different with regard to the serous membranes. The development of a multi-layered "epithelium" from the serosa is well documented. Areas of stratification occur in various parts of the peritoneum, particularly in relation to the Fallopian tubes and broad ligament. These are often referred to as "Walthard nests." Many of the structures included under this term are complex and different from the simple stratification under discussion. Cellular accumulations with cyst formation (Heinz, 1953) are common and seem to be metaplastic variants of simple swelling and stratification of serosal cells. Although most attention has been directed to Walthard's paper (1903), as with many other conditions, clear descriptions of the phenomenon had been given earlier. The earliest account appears to have been one by Werth (1887). Following this numerous examples were recorded (Ries, 1897; Rossa, 1898; Pick, 1901; Schickele, 1902; amongst others).

Though the phenomenon has been observed more often in the female, an example in the male was reported by Meyer in 1903. Since then many examples in the female pelvis have been recorded both in young people (Akagi, 1928) and in older individuals (Danforth, 1942). A description of changes, experimentally produced, in the pleural membrane was given by Young in 1928.

Descriptions of this stratification in the peritoneum were also given by Reis (1946) and Greene, Peckham and Gardner (1949), but these authors considered that there was no evidence of squamous change. Similar conclusions were arrived at by Teoh (1954).

Examples of the same kind of change were demonstrated in the coverings of the epididymis and in the adjacent tunica by Hartz (1947) and Sundarasivarao (1954). Excellent demonstrations of the phenomenon are given in this second paper. Though obviously they illustrate a stratified epithelium, these writers considered that it was not squamous in type and discounted the possibility of such an occurrence.

On the other hand Crome (1950) described similar cellular collections on the peritoneal surface of the intestine but, because of

the presence of demonstrable intercellular prickles, regarded the epithelium as being squamous in form. The writer has recorded indubitable squamous epithelium in encysted hydroceles of the cord (King, 1951). In one case keratin, demonstrable both on morphological and histochemical grounds, was present.

Thus from the present point of view, this development of "epithelium" from an internal lining—usually referred to as a mesothelium rather than epithelium — is now thoroughly substantiated; here not only does stratification occur but also a squamoid or even squamous differentiation is occasionally to be seen. It is apparent from what has been said above that many writers have not agreed that squamous change occurs; and this is true of the cases described by them. Stratification is indubitable but the necessary criteria for the demonstration of squamous change have not been present. There is some glycogen in many of the cells and often the intercellular prickles are irregular and by many are regarded as artefacts. Such interpretation is quite reasonable in most of the cases but examples of more extreme change are observed; in these the morphological similarity to typical squamous epithelium of various kinds, the typical appearance of keratin laminae and histochemical demonstration corresponding with these provide sufficiently precise criteria for recognition of the nature of the epithelium.

This is an infrequent observation which, of course, indicates that the stimulus necessary for the production of this kind of epithelium is, as might be expected, infrequently effective in this situation. It is important to appreciate that negative observations must be accepted with some caution especially when a condition has not been observed with great frequency. There are many examples of changes which were not observed or recognized until a large number of cases had been collected; thus until the absence of a phenomenon has been demonstrated a large number of times it is unwise to assume that it does not occur. Moreover one positive observation is worth more than all the negative ones that can be collected. As was remarked earlier a very important method of advance in modern times is the accumulation of large numbers

of cases. When experimental production of a condition is possible problems may be solved relatively easily but when this is not practicable, that is to say, when we do not know the stimuli which give rise to a particular condition it is then necessary to wait for the result of the "natural experiment." In several fields this simple accumulation of information has demonstrated both new conditions and variants of previously known ones which had not been suspected and, in some cases, had been thought impossible.

In the tunica vaginalis, almost invariably —that is to say normally—the lining is that of a single layer of mesothelial cells. When this is damaged it is often completely destroyed and it is replaced by a granulation tissue which merges into necrotic material. However, the uncommon example of proliferation and metaplasia of the lining, even though rarely observed, is important as showing that a capacity for change of this type is not confined to the peritoneum proper. The metaplastic change in the serosa usually is not seen when there is a grossly thickened tunica but rather when the tissues are relatively thin and macroscopically normal. This suggests that the stimuli producing gross chronic inflammatory changes in the region are not those which induce these metaplastic changes in the serosa.

A change in connective tissue linings, of the synovial type, need not detain us at present though examination of some synovial cysts, bursae or cysts of the lateral meniscus of the knee will provide examples of stratification and a morphologically definite epithelium is to be observed in some of the examples of synovial tumour. However, here, the discussion is confined to epithelia of serosal origin.

The nature of the stimulus causing the changes is unknown. It is quite clear that, in some epithelia, exteriorization is important, as for example in the polyp of the cervix uteri or a haemorrhoid; on the other hand pus retention, if the epithelium is not completely destroyed, gives gross changes as, for example, in *kraurosis* or *ichthyosis uteri*. In the case of the serosa such gross changes in the environment of the cells result in their death. It is most probable that several factors

play a part and the distribution of this kind of change suggests that one of these is hormonal in type.

In the present example there had been a chronic inflammation in the area involving the epididymis but there was no suggestion that this was significantly different from what occurs in many other cases in which a hydrocele of the tunica vaginalis accompanies an epididymitis.

In the present case, the importance of the phenomenon is the demonstration of the capacity of tissue cells, in still one other part of the body, for the production of a stratified and squamous epithelium. The general importance of this lies in the demonstration that this type of epithelium can occur in any part of the body, in adult life, developing from the local tissues (even mesodermal and not what is ordinarily regarded as "epithelium") so that when it is seen in abnormalities arising late in life or in tumours, for example, in some of the "teratomata" it should be quite apparent that the presence of squamous epithelium, *per se*, is no evidence at all of any significant complexity of structure. It merely indicates one of the changes that may occur in any type of epithelium or mesothelium.

SUMMARY

1. An example of epithelial transformation of the serous lining of the tunica vaginalis testis is described.

2. The epithelium is stratified and, in parts, both morphologically and histochemically is squamous in type.

3. The literature relating to the problem of this kind of metaplasia in serous membranes is reviewed.

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RENAL AGENESIS AND UNICORNUATE UTERUS

REPORT OF A CASE AND AN ANALYSIS OF ITS DEVELOPMENT

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THE observation that maternal rubella has a detrimental effect on the embryo (Gregg, 1941) has sharpened interest in the association of maternal disease and foetal abnormality.

It is intended here to record a malformation — unilateral renal agenesis with unicornuate uterus noted as an incidental finding at autopsy — and to analyze the underlying defective tissue organization which resulted in this gross malformation. By such an analysis it is possible to dissect the mechanics of a malformation though the precise nature of its pathogenesis still remains a mystery.

CASE REPORT

C.W., a single girl, aged 18, had been born and reared in Melbourne. She had suffered no serious illness in the past, neither had she had any symptoms referable to her urinary or genital tracts. Menses had been regular lasting 5 days. She was admitted to hospital with abdominal pain, for which laparotomy was performed, and subsequently she succumbed to a complication of the operation not related to the present findings.

FIG. I. Photograph of the genital organs from the antero-superior aspect showing:

(a) Absence of the right Fallopian tube;
(b) absence of the right broad ligament;
(c) large right ovary;
(d) right round ligament attaching to the cervix.

At autopsy a gross malformation of the genito-urinary system was an incidental finding. The right kidney was completely absent (Fig. III). There was no renal artery or vein; the right adrenal gland

4A.

was present and normal in its size and situation. The left kidney weighed 5 oz. and was quite normal, as was the left ureter; the right ureter was absent.

The uterine fundus was anteverted and deviated to the left side of the pelvis by approximately 20 degrees although the cervix uteri was in the midline (Fig. I). The uterus measured 4.5 cm. by 2½ cm. by 2½ cm. and the fundus, instead of being round, was pointed and the left Fallopian tube was attached to the apex. On the left side, the broad ligament, Fallopian tube, round ligament and ovarian ligament were normal. On the right side, there was no Fallopian tube. The ovary was large and situated near the internal inguinal ring. It was 5 cm. by 2 cm. by 1.8 cm. compared with that of the left side, 3½ cm. by 3½ cm. by 2 cm.
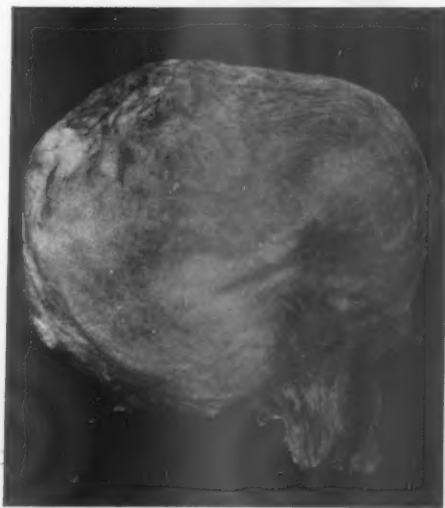

FIG. II. Photograph of the bladder trigone showing:

(a) Atrophy of the right half of the trigone;
(b) absent right ureteric orifice.

Attached to the upper pole of the right ovary was the round ligament, which ran retroperitoneally between the ovary and the junction of body of uterus and cervix where it was firmly attached. There were about 3 cm. of poorly formed tube attached to posterior surface of the ovary. The broad ligament was absent. External genitalia, vagina, cervix uteri were normal.

The right ureteric orifice into the bladder was absent; that of the left side was normal in size and position (Fig. II). There was atrophy of the right half of the trigone; the urethra was normal. The pelvic vessels were not dissected. No other malformations were observed at autopsy.
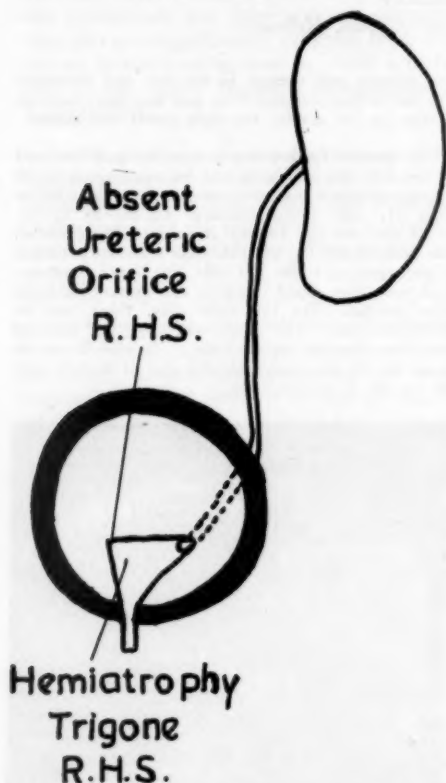

Absent
Ureteric
Orifice
R.H.S.

Hemiatrophy
Trigone
R.H.S.

This is the thirty-first reported case of what must therefore be considered an uncommon malformation. (For reviews of former cases see Anders, 1910; Schumaher, 1938; Daro and Collins, 1950.)

It is of embryological interest and clinical importance to note that both the urinary and genital systems are affected.
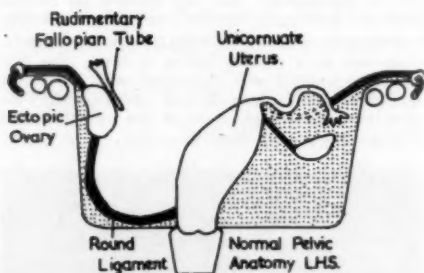
If a malformation is found in the genital tract, is it likely to be accompanied by renal agenesis? Anders (1910) found that renal agenesis accompanied genital malformation in 90 per cent. of 135 cases. Meredith

Campbell (1951) estimated that the upper urinary tract was anomalous in about one-third of cases of major genital malformation.

Conversely, if renal agenesis existed, Schumaher (1938) estimated that 70 per cent. have an associated major genital malformation. Anders, in a review of 61 cases of unilateral renal agenesis, found that 14 had some major genital malformation.

In spite of the wide discrepancy in the figures, it would seem clear that malformations in one of the two systems are accompanied, in quite a significant proportion of cases (at least one-third) by anomalous development in the other.

Meredith Campbell made the statement: "embryologically and anatomically, the urinary and genital tracts must be considered as a single system." A more detailed consideration first of the normal embryology and then of the development of the malformation described above will illustrate how interdependent the two systems are.

Rudimentary
Fallopian Tube   Unicornuate
                        Uterus.

Ectopic
Ovary

Round   Normal Pelvic
Ligament   Anatomy LHS.

FIG. III. A schematic representation of the genito-urinary organs.

NORMAL DEVELOPMENT OF THE UROGENITAL SYSTEM

The intermediate cell mass lies on the dorsal wall of the embryo between the paraxial and the lateral plate mesoderm. In it develop the pronephros, mesonephros, metanephros, gonad and pronephric or Wolffian duct.

About the second to third week of intrauterine life the pronephros appears and its duct grows caudally by division of apical cells to reach the cloaca at about three to four weeks. By this time, the pronephros has degenerated and the mesonephros appears, growing distally with the Wolffian duct.

The gonad develops on the median side of the urogenital ridge at the fifth to sixth week and also, at this time, the Müllerian duct is formed on the lateral side of the ridge and the Wolffian duct, possibly by invagination of coelomic epithelium (Fig. IV).
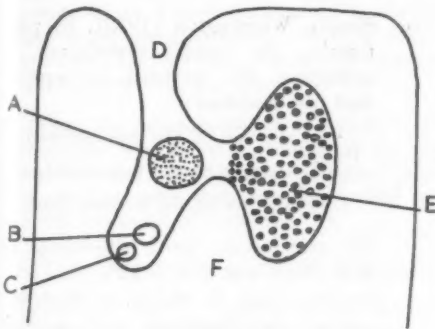

FIG. IV. Diagram of a transverse section through a twenty-five millimetre embryo showing the components of the urogenital ridge on the dorsal wall coelomic cavity.

A. Degenerating mesonephros.
B. Wolffian duct.
C. Müllerian duct.
D. Urogenital mesentery.
E. Gonad.
F. Coelomic cavity.

The ureteric bud, a dorsal outgrowth of the Wolffian duct makes its appearance in the fourth to fifth week and reaches the most caudal part of the urogenital ridge, the future metanephros, about the fifth to sixth week. When this occurs, the metanephros (future functional kidney) differentiates.

Thus, by the end of the sixth week, in normal development, the pronephros has matured, degenerated, and its duct, the Wolffian duct, has

(a) entered the cloaca,

(b) budded off the ureteric bud which appears to be responsible for metanephric development, and

(c) provided an excretory duct for the mesonephros.

About this time the Müllerian ducts are growing downwards on the lateral side of the Wolffian duct — actually between the mucous membrane and basement membrane of the Wolffian duct — growing by division of the apical cells of the Müllerian duct. At the pelvic brim the duct leaves the Wolffian duct, crossing ventral to it to enter the urogenital sinus (Fig. V).
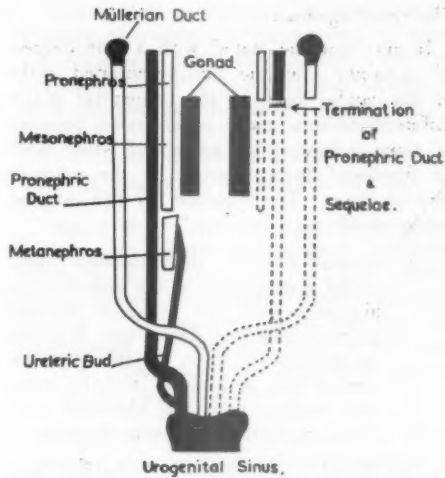

FIG. V. A diagram showing the development of the urogenital system. Those parts dependent on the Wolffian duct or its outgrowths for organisation are shown in white. On the right-hand side the results of sections of the Wolffian duct at the point indicated are shown schematically.

The rudimentary uterine ligament is found in the peritoneal fold running from the dorsal urogenital ridge to the groin on the ventral surface of the embryo. This tissue pierces the inguinal canal and attaches to the site of the future labium major. It thus links gonad, Müllerian duct and the future labium major.

EMBRYOLOGY OF THE MALFORMATION

In the case under discussion the following anomalies require explanation:—

(1) Right renal agenesis;

(2) absent right ureter;

(3) hemiatrophy of the trigone on the right side;

(4) almost complete absence of the right Fallopian tube;

(5) unicornuate uterus;

(6) abnormal attachment of the round ligament;

(7) an ectopic ovary on the right side.

The development of each of these will be discussed in turn. Following this, the

position of a single lesion capable of producing all the above anomalies will be considered.

The renal agenesis

It may now be stated with a fair degree of certainty that the undifferentiated cells of the caudal half of the urogenital ridge differentiate into renal parenchyma because of some influence, the nature of which will be discussed later, exerted by the ureteric bud on them. The evidence for this may be summarized:—

(a) Brown (1931) demonstrated that, whilst a deficiency in the metanephric blastema did not prevent normal ureteric budding from the Wolffian or pronephric duct, it did seem essential for a ureteric bud to come into contact with the blastema mass for a functional kidney to develop.

(b) Renal tissue has very rarely been described as a component of teratomata. This would appear to be a fact requiring some explanation in view of the variety of tissues generally obtained here.

Nicholson (1950), in 1922, said: "Renal tissue has not hitherto been described in teratomata. I have not been able to satisfy myself that the secretary part of the corresponding metanephros is ever developed from the intermediate cell mass when there is complete aplasia of the ureteric bud of the Wolffian duct. It remains to be seen how far the development of the ureter and kidney are correlated with each other, and if the former acts in some way as a stimulus to the other."

However, in 1934, after describing a teratoma possessing functionally and histologically differentiated metanephric tissue adjacent to a cyst lined by transitional epithelium, he states: "No secreting kidney is known in the primary absence of collecting tubules. I submit that the transitional epithelium is the morphological and physiological homologue of the somatic ureter." Thus, it may be concluded that, in teratomata, without ureteric bud or its homologue, there is no differentiation of primitive embryonic blastema into metanephric tissue. This infers that the differentiation of metanephros is dependent on some organizing action of the ureteric bud.

(c) Finally, Waddington (1938), by performing the crucial experiment of sectioning the growing pronephric duct has produced:—

    (i) absence of the mesonephros;

    (ii) absence of the metanephros;

    (iii) absence of the ureteric bud.

His conclusion, worth repeating, is that "this vestigial organ, the pronephros, has, in reality, a most important morphogenetic role to play in the development of the urogenital system."

Boyden (1927) and O'Connor *et alii* (1938) have performed similar experiments in birds, confirming Waddington's results in amphibia although the conclusions drawn were not identical.

The absence of the right ureter and right trigonal atrophy follow à *priori* from the above evidence.

There would thus appear to be adequate evidence that without a ureteric bud prospective embryonic renal tissue does not organize into a morphological kidney.

Genital anomalies

Gruenwald (1949) has performed experiments similar to those described by Waddington but has made the additional observation that, if the growing Wolffian duct be severed, almost complete absence of the Fallopian tube and unicornuate uterus accompany the renal agenesis on the affected side (Fig. V). The growing Müllerian duct is completely dependent on the Wolffian duct, either for guidance or for a supply of cells and, should the growth of the pronephric duct be interrupted at a point, then that of the Müllerian duct also ceases at this point. Anomalies identical with those of this case have been produced in chick embryos by this means.

The remaining anomalies, ectopic ovary and abnormal attachment of the round ligament, would appear to be the result of purely mechanical forces. The round ligament presumably has remained free for a greater length of time and has consequently been able to pull the ovary further down into the pelvis before it becomes attached to the first part of the uterus it meets—the only midline structure, the cervix, originating from the Fallopian tube of the other side.

Thus, the development of almost the entire urogenital system is dependent on the pronephros and its duct; a vestigial organ functioning only between the second and fourth weeks of intra-uterine life.

THE MORPHOGENESIS OF THE MALFORMATION

Evidence has been presented in the preceding paragraphs that the metanephros, the adult kidney, and the Müllerian duct, and the adult Fallopian tube and uterus, are dependent for their development on the presence and integrity of the Wolffian or pronephric duct.

Should Wolffian duct growth be interrupted in the lower part of its course, there will be absence of the metanephros. The mesonephros, in addition, will be absent if the disturbance to the duct be a very high one.

A malformation very similar to this one would have resulted if the pronephros, itself, had failed to differentiate. However, such an occurrence would be accompanied in all probability by other developmental anomalies outside the genitourinary tract. As this malformation was a solitary anomaly, pronephric agenesis is not considered to be a likely cause for the case under discussion.

As it is not known whether there were any remaining mesonephric rudiments, all that can be said is that the anomalies described were due to some interruption of growth of the Wolffian duct between the pronephros and the ureteric bud, or temporally, between the fourth and sixth weeks of intra-uterine life.

This malformation illustrates two important principles:—

1. The interdependence of organs in development.

2. The possibility of widespread results from very small foci of abnormal development in the foetus.

It would seem necessary, in conclusion, to attempt to summarize the notions of tissue organization in order that the theoretical and perhaps eventually practical implications of such analyses as this will be appreciated.

THE PHYSIOLOGY OF ORGANIZATION AND THE PATHOGENESIS OF MALFORMATION

"Embryology over the past 50 years has become an analytical science studying developmental mechanics" (Weiss, 1939). Simple growth will produce mass, differential growth will produce shape, cellular differentiation will produce tissues; but how is this conglomeration organized into a definite and typical pattern?

To attempt to give an answer to these questions, the experimental method must be employed. Amphibian embryos provide the main material. It will be recalled that the egg is fertilized, segments into the blastula and then becomes a gastrula, characterized by invagination in the vicinity of the grey crescent and differentiation into primary germ layers, primitive gut roof, notocord, myotomes, etc. Next, the neurula is recognized, having true neural folds.

Here, then, is a period of intense embryonic activity — nondescript cells are being organized into tissues and organs—cells with multiple potentialities are being directed in particular directions; primitive embryonic blastema is becoming nervous tissue, renal tissue or perhaps limb bud.

Dreitch (1929) first pointed out this fact and introduced the terms "prospective significance" and "prospective potentiality," the former indicating the actual fate of a cell and the latter to signify the possible fate of such a cell.

The prospective potency of a cell is much greater than its prospective significance up to a certain point in its life, after which development proceeds along a certain line. This change in potency is called "determination." Up to the stage of mid-gastrulation the cell is undetermined; it is still pluripotent. In late gastrulation its fate appears

to become irrevocably determined — "prospective potentiality has been ruthlessly curtailed for prospective significance" (Needham, 1942).

In the field of organogenesis, this notion of limitation of cell potentiality during organization is a necessary one, but the possible participation of these "irrevocably determined" cells in the changes of metaplasia and neoplasia during later life necessitates the postulate of a return of the former cellular plasticity at such times.

There are a whole host of organizers controlling development and there exists, among them, a certain order in time as well as space. Secondary organizers are dependent on the tissues which the primary organizer has induced, tertiary organizers on the tissues which secondary organizers have induced.

The optic lens is formed by the organization of head ectoderm when the optic cup contacts it. The optic cup contains a secondary organizer. Should the optic cup be damaged before it reaches the ectoderm, the lens fails to develop (Spemann, 1901), although the lens forming ectoderm is not injured by the operation affecting the optic cup.

Numerous investigators found evocators, or organizers, for various induced structures and there results a "hierarchy of organizers" (Needham, 1942).

So far as the urogenital system is concerned, it appears that mesoderm induces pronephros, acting as a secondary organizer. Pronephric duct induces mesonephros and cloacal diverticulum, acting as a third grade organizer. Ureteric bud induces metanephros, acting as a fourth grade organizer.

The defect in the case presented appears to be at the level of the third and fourth grade organizers. There are numerous and obvious gaps in present knowledge—control of evocation and the precise nature of cell competence being of particular importance. It is known that there is some genetic control of evocation and also that changes in environment, such as Vitamin A deficiency, short wave irradiation, intrauterine anoxia will impair the induction of tissues. However, there is very little concrete knowledge of the subject at the moment.

All that can be said is that, should tissue and organ induction be impaired at this critical period of growth, even for a very short time, large malformations can result.

The particular organ affected depends on the time of application of the stimulus since different systems are undergoing individuation at different times.

SUMMARY

A rare malformation has been reported—renal agenesis in association with unicornuate uterus.

The mechanics of its development have been discussed emphasizing the embryological interdependence of the urinary and genital systems. The relation of this interdependence to "tissue organizers" has been analysed.

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RESTORATION OF PALATAL AND LIP DEFECTS

By H. P. PICKERILL

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OCCASIONALLY palatal losses or defects are successfully closed by a prosthesis, but when the patient is edentulous it usually fails. There then remain three other surgical means of closing the communication between the mouth and nose or antrum. The first method, and one nearly always successful for smaller openings, is the raising of a sufficiently long and wide flap from the buccal sulcus, lining it with a razor graft and at a second operation suturing it very carefully with fine silk sutures to the well rawed margins of the orifice. The patient must be warned to avoid sneezing or whistling and must wash his mouth out thoroughly, but gently, after every meal for fourteen days. The writer has routinely closed many such defects, usually those due to portions of alveolar process (the maxillary tuberosity) having come away during the extractions of molar teeth. I have never seen a failure. For larger losses or defects such as gun-shot wounds, accidents and wide inoperable cleft palates in adolescents, the tube-graft undoubtedly gives the most satisfactory results. The graft, in order to get the most suitable skin and the most advantageous position, should be constructed along the clavicle. I long ago abandoned the neck tube-graft. Its disadvantages are that it nearly always leaves an ugly and perhaps keloid vertical scar in the neck, and in the male may cause hair bearing skin to be imported into the mouth.

In female patients when there is an objection to even a much finer scar along the clavicle; the tube may be made on the abdomen and by attaching one end to the wrist be thus carried up to the mouth, lip or nose. The tube-graft having been constructed and matured, the medial end of the tube preferably is divided and attached to the inner surface of the upper lip which affords a full blood supply very quickly. A fortnight later the shoulder end of the tube is detached and the tube shortened to the necessary length.

The margins of the defect are thoroughly excised, lateral incisions made on the tube at its distal end and secured to the rawed orifice or cleft by a double row of sutures— the deep ones 6-0 white silk and the superficial 1-0 black silk. This ensures satisfactory approximation, which must be exact and absolute and without any tension. In fourteen days the unwanted portion of the tube can be excised and discarded and a few additional sutures inserted in the proximal end of the tube and orifice. This affords a very sound repair over which an edentulous patient can wear a denture with plenty of "suction" and therefore be very comfortable and can eat and speak normally. The original of this method (now slightly modified) I described in the *British Journal of Surgery* in 1922 (see Fig. I), in my text book on facial surgery in 1924 and also in the *Medical Journal of Australia* in 1928. The method was described also by Balcombe Quick in 1929— unaware apparently of earlier descriptions. The latter author has now described a third method, namely by suturing the tip of the tongue into the orifice or defect. This has the advantage of two operations instead of four.

One of the criticisms of the tube-graft method of restoration was that it entailed much pain and discomfort to the patient (as a matter of fact it entails none if done as described above), but one would think it would be difficult to avoid such criticism of the tongue method.

There is one thing which is absolutely essential in whatever method is used, namely the full co-operation of the patient. Every detail of what is going to be done must be explained to him, and it is advisable to obtain a written statement from him that he thoroughly understands what is going to be done and that he will co-operate and help to his utmost. No restrictions when using the tube method need be placed on talking or

feeding, except that the latter must be fluid or very soft, milk and milk puddings are good—broths and soups are banned, at least for the first week after the tube is in the mouth, for the reason that the product of fermentation of milk is lactic acid — a good antiseptic, whilst soups promote the increase of proteolytic and suppurative organisms which may destroy or prevent union. I have no experience of the tongue method but restrictions on speech and feeding one would think would be severe and the co-operation of the patient a little difficult.

there is no muscle action remaining, function is naturally not so good and shrinkage of the graft occurs. Nevertheless patients are always gratified to be able to abandon an "obturator." The same method of tube-graft restoration of the upper or lower lip (or both) after much wider excision for epithelioma than is possible if the wound is to be closed by approximation, is also described in the *British Journal of Surgery*, 1922, and has been frequently and very successfully used by the present writer during the intervening years (see Fig. II). It can be recom-

(a)   (b)   (c)

FIG. I. (a) Plaster cast of palatal defect suitable for restoration by means of a tube-graft. Jan. 6, 1920.

(b) Tube-graft fashioned from the neck, and passed in through the mouth to remedy the defect shown in (a). The lips were sutured together temporarily to prevent movement. No very efficient antiseptic precautions were possible, yet the graft "took" perfectly. Jan. 23, 1920.

(c) Plaster cast of restoration by tube-graft of defect shown in (a). Feb. 16, 1920.

(Reproduced by permission of the Editor of the "British Journal of Surgery" in which these illustrations appeared in 1922.)

The best results, by the tube-graft method in those neglected wide clefts of the hard and soft palates, are obtained in those cases in which there is some muscular action still present in the soft palate. Good function and speech can then be anticipated. One such cleft palate patient, operated on when a boy, by this method many years ago has now become such a good speaker that he is the secretary of a trade union and argues their cases in court very effectively. In those cases where there has been considerable loss of tissue due to infection and sloughing and

mended as giving much better functional and cosmetic results than the approximation method, not only so but it eliminates the tendency to limit the extent of excision because of the fear that approximation may become impossible. It takes longer but it may save life. It is always better to be certain than quick.

In all tube-graft restorations of palate or lip local anaesthesia by the Corlette method has been used—except that omnopon has been given instead of morphine and sometimes heroin only in the second or third dose—
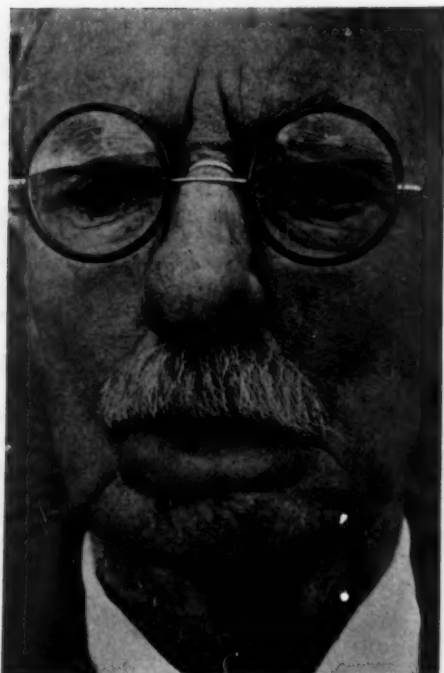
FIG. II. Restoration of lower lip by "clavicular" tube-graft after total excision for recurrent epithelioma (resistant to radiation), six years ago. The new lip is fully functional, sensitive and pink in colour. He wears a full lower denture with comfort, smokes cigarettes without burning his lip and there is no sign of recurrence.

often on the table. This ensures a dry field of operation with no obstructing anaesthetic tubes and much less haemorrhage, a placid patient, less risk of post-operative vomiting and quicker co-operation by the patient on return to the ward.

Note

At Queen Mary's Hospital for facial wounds at Sidcup during World War I, we all felt very proud of ourselves when using and demonstrating tube-grafts as being a great advance in plastic surgery. Now, however, we know that they were used by Gaspare Tagliacozzi (1545-1599). This Italian surgeon raised a long flap of skin from the upper arm (which possibly tubed itself) for his rhinoplasty operation. The illustrations in his book make this quite clear. The method, like many another good thing, would appear to have been dormant for a long time, in this case for nearly 400 years.

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A NEW WALKING CALIPER

By Peter Williams
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IN 1875 Hugh Owen Thomas first described the walking caliper which now bears his name. The caliper has stood the test of time, and is accepted as a most valuable appliance in the treatment of a wide variety of disabilities. Although originally used chiefly in the after care of fracture and of tuberculosis of the knee, it has come to be used for many other purposes, and especially for paralytic conditions of the lower extremities. It is particularly in the latter instance that the Thomas caliper is known to have some serious disadvantages, not the least of which are its weight and bulk and the awkward gait it produces. These disadvantages become more apparent in children, and at any age when calipers are worn on both legs.

In an effort to overcome these problems, experiments have been carried out over the past two years, and after many trials and tribulations, the caliper to be described has been evolved, and has proved extremely satisfactory both to the patient and to the surgeon. It has proved lighter in weight, more comfortable and more easily applied by the patient, and, owing to the greater freedom of ankle movement allowed, the gait is correspondingly improved. The mechanics of the design are such that drop foot stops or springs are unnecessary, for the tension on the knee strap holds the foot at right angles to the leg. When worn in pairs, the calipers do not strike each other on walking, and in women the caliper is not visible from the front.
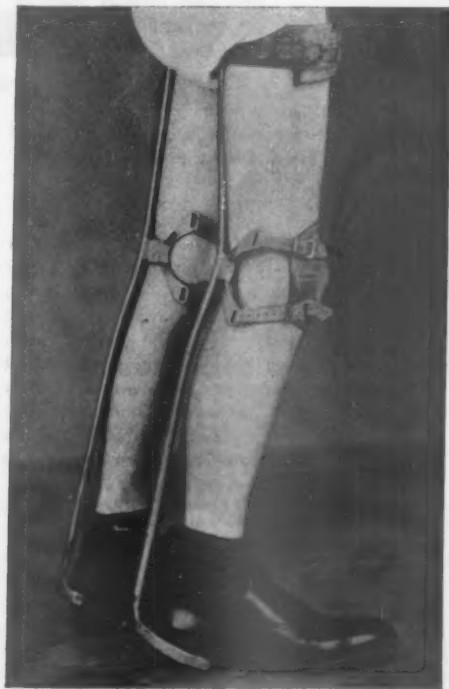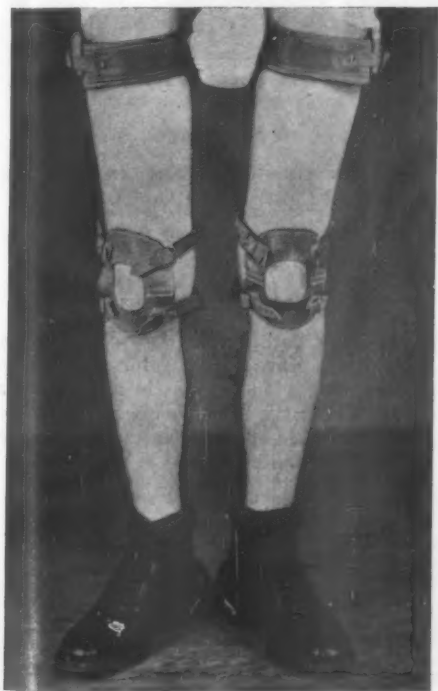

FIG. I

Experience has shown that this caliper is unsuitable if any significant degree of knock-knee is present, or if T-straps are necessary to control ankle instability.

The greatest field of usefulness appears to be in poliomyelitis, and in the management of permanent paraplegia following spinal injuries. It has a limited application as a

was constructed, as illustrated in the accompanying clinical photograph (Fig. II).

A tubing of 16 gauge and ⅝ inch in diameter is used for the bar of the adult caliper (Fig. I). For the child's caliper, hard drawn carbon wire is used, the diameter varying from ¼ inch to ⁵⁄₁₆ inch, according to the age. Bronze welding is preferable for attaching the knee and top plates.

FIG. II

knee brace after patellectomy, in rheumatoid arthritis and in sundry other conditions.

CONSTRUCTION

The caliper consists of a single posterior bar surmounted by a band top, and articulating with the shoe via a U-shaped stirrup. The knee is secured to the caliper with the conventional leather cap and four straps. For reasons which are not fully apparent, it has been found necessary to construct slightly different designs for adults and children: repeated breakages at the lower end in children were finally solved when the stirrup

SUMMARY

1. A new type of walking knee brace is described.

2. Certain advantages over the conventional caliper are suggested.

AN UNUSUAL CASE OF THROMBOPHLEBITIS AND A NEW TYPE OF VENOGRAM

By M. F. A. WOODRUFF

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THE following case presents some unusual features and illustrates the use of a new technique for obtaining venograms recently introduced by Dr. Charles Begg (Begg, 1954).

CASE REPORT

The patient, a single girl aged 26, was admitted to the Dunedin Public Hospital on 9th July, 1953.

History

In 1944 she sustained a simple fracture of the middle third of the right femur. Following this the right knee became stiff and painful, and in 1948 it was arthrodesed. A few days after this operation the patient developed femoral thrombosis on the sound (left) side. This was treated effectively with heparin and dicoumarol, and subsequently the left leg caused little trouble until March, 1951, when the patient had an attack of thrombophlebitis, characterized by swelling and tenderness of the limb, and pyrexia. She was treated with anticoagulants and penicillin for three weeks without effect, but improved later when given aureomycin.

In September, 1952, the patient had a further attack of thrombophlebitis of the left leg which failed to respond to conservative measures and was eventually treated by lumbar sympathectomy. The condition subsided but the leg became swollen and painful once more in January, 1953, and again in July, 1953.

Clinical findings

When first seen by the writer in July, 1953, the subject of this rather melancholy history was in a state of acute anxiety because she had been told that pulmonary embolism might occur at any time.

On examination the temperature was 101° F. and the pulse rate 110. The left leg was swollen and tender below the knee, and there was pitting oedema at the ankle. The colour of the limb was normal. The patient complained of pain in the left iliac fossa and there was tenderness on deep palpation in this region.

Treatment with anticoagulants and antibiotics (penicillin followed by terramycin) produced no change and the patient's temperature continued to swing between 99° and 101.3° F.

On 18th August a venogram ws done by Dr. Charles Begg by the following technique:—

A special trocar and cannula, approximately the diameter of a 17 gauge needle, was introduced into the great trochanter of the left femur. Twenty ml.

of 50 per cent. uriodone was then quickly injected through the cannula and two A.P. films were taken, one as soon as all the medium was injected and the other about four seconds thereafter.

FIG. I. Venogram showing obstruction of the left common iliac vein, with large collateral venous channels joining the right and left internal iliac veins.

The venograms thus obtained were far superior to any others the writer has seen. They showed that the left common iliac vein was completely obliterated, and that venous return from the left lower limb was dependent on large venous channels linking the two internal iliac veins.

It was decided to ligate the left common iliac vein at its junction with the vena cava, principally as a prophylactic measure against upward extension of the clot. The operation was performed on 20th August. The left common iliac vein was exposed

by a transperitoneal approach. It was found to be narrowed, and filled throughout its length with firm, and apparently well-organized, clot. The vein was ligated in continuity at its junction with the vena cava.

After the operation the temperature continued to swing to 100° F. for about four weeks and then became normal. The patient returned to her home town and has not been seen here again, but her doctor reports that she is back at work as a hospital dietitian and has had no further symptoms.

COMMENT

The main purpose of this report is to draw attention to a new method of venography, and to the extraordinary rapidity with which material injected into cancellous bone enters the circulation.

With proper precautions the method is safe and yields excellent results. It is being used mainly to locate incompetent communicating veins in patients with chronic ulcers

of the leg, and a report of this work will be published later.

The indications for vein ligation are much disputed, and it is not intended to discuss them here. Suffice it to say that in the case reported it seemed clear that there was nothing to lose by ligating the common iliac vein, and the present indication is that much has been gained.

SUMMARY

A case of thrombosis of the left common iliac vein is described in which the diagnosis was confirmed by a new type of venogram. The condition was treated by ligating the affected vein at its junction with the inferior vena cava.

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CARTILAGE AND BONE FORMATION IN FIBROADENOMATA OF THE BREAST

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THE occurrence of cartilage and bone in human breast tumours is an extremely rare phenomenon. Rottino and Willson (1945) reviewed the world literature from the year 1700 and found reports of only 36 mammary tumours containing cartilage and/or bone. In the present paper 2 new cases are described to introduce a discussion of the histogenesis of cartilage and bone in epithelial tumours.

HISTORICAL

Bonet (1700) may have been the first to record a bony mammary tumour when he described a tumour "which could not be cut with a knife." Rottino and Willson (1945) found that since the advent of the microscope there have been reported (to use their classification) 7 enchondromata, 8 osteochondromata, 2 mixed tumours containing cartilage, one teratoma containing cartilage and one chondrolipoma. The remainder of their total of 36 was composed of malignant tumours. There was no record of an enchondroma other than in the French, German and Italian literature and none since 1909. Furthermore records of osteochondromata, apart from one each from France and the United States, were confined to the British and German journals, the latest report having been made in 1929. Rottino and Willson claimed that a fibroadenoma had been the parent tumour in one osteochondroma and one enchondroma, but suggested that, had they been examined at an earlier stage of growth, others would have shown a parent fibroadenoma. To this list must be added 2 cases of cartilagenous transformation in fibroadenomata reported by Cheatle and Cutler (1931) and also a pure cancellous osteoma of the breast described by Robinson and Spencer (1950).

In contrast to the rarity of occurrence in human breast tumours, cartilage and bone have been found often in mammary tumours in bitches and rats. Glendining (1910) recorded cartilage and/or bone in 4 of 8 benign tumours of dogs. Allen (1940) described 4 malignant tumours containing cartilage and bone among 13 mammary tumours examined from dogs and a later report by Willis (1953) described cartilage and/or bone in 6 of 17 canine mammary tumours.

FIG. I. Photomicrograph of Case 1 showing the edge of the intracanalicular fibroadenoma. On the right the epithelium has disappeared, leaving clefts in an otherwise homogeneous hyaline stroma. This stroma has become calcified as seen in the lower right corner. (x 30)

CASE HISTORIES

Case 1

Clinical history. The patient, a female aged 45 years, presented with hard nodules in one breast. The history and results of clinical examination, apart from these observations, were not available.

Pathological examination. There were multiple small lumps in the breast which, in due course, were shown to have the histological structure of intracanalicular fibroadenomata. One of these claimed special attention—a discrete nodule 2.5 cm. in diameter. Histologically it was enclosed in a fibrous connective tissue capsule blending with the peripheral mass of the tumour which presented an overall lobular pattern with the structure of an intracanalicular fibroadenoma. A few peripheral lobules were typical with well-formed cuboidal epithelium and mucoid and fibrous stroma. Nearer the middle of the tumour the epithelial layer was thinner and in places had disappeared while the stroma had become almost acellular and homogeneous. A further stage in this change had given rise to clefts, from which the epithelium had gone, separating masses of eosinophilic hyaline material. The arrangement of these masses of tissue still retained the architecture of the original fibroadenoma. Large areas of this material had become calcified and merged with foci of bone (Fig. I). The bone was composed of moderately cellular thin trabeculae separted by a marrow made up of fat and thin-walled blood vessels, but no haemopoietic tissue (Fig. II). There was no epithelium in contact with the bone nor was any cartilage seen in the section.

FIG. II. Photomicrograph of Case 1 showing bone trabeculae separated by a marrow of fat and blood vessels. A tongue of fibrous stroma containing epithelium can be seen projecting from the bottom of the section. (x 60)

Diagnosis. The diagnosis of intracanalicular fibroadenoma of the breast with calcification and ossification of the stroma was made.

Case 2

Clinical history. Mrs. M. O'M., aged 62 years, a married woman with five children, was admitted to the Royal Melbourne Hospital on 10th February, 1954, complaining of a lump in the left breast present for two years and accompanied by a creamy nipple discharge and, on only one occasion three months prior to admission, bleeding from the nipple. She had noticed moderate aching pain in the breast and had lost 7 lb. in weight since the episode of bleeding. Examination revealed a hard, mobile lump 1.5 cm. in diameter deep to the edge of the areola and inferior to the nipple. There was no attachment to any structure other than the nipple and no discharge could be expressed. The lump was removed with some surrounding breast tissue on 11th February and the patient was discharged from hospital on the following day. Convalescence was uneventful.
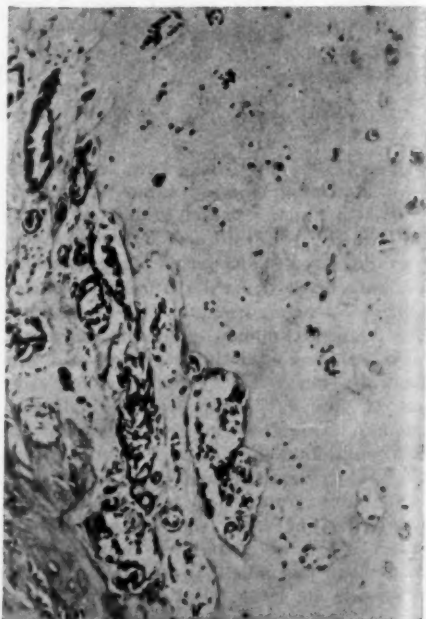

FIG. III. Photomicrograph of Case 2 showing the region of junction between the cartilagenous portion and the remainder of the tumour. The capsule can be seen in the lower left corner. The specimen is poorly fixed and there is considerable epithelial distortion. (x 80)

Pathological examination. The lump was found to consist of two round hard nodules of one cm. and 4 mm. in diameter respectively. The larger projected in polypoid fashion into a dilated duct invaginating its wall from the outside. On section the nodules were glistening white in colour with the consistency of cartilage, shattering when compressed with forceps; translucent flakes could be cut from

the surface. The dilated duct and others in the vicinity contained a yellow-brown creamy material, but there was no unchanged blood.

FIG. IV. Photomicrograph of Case 2 showing another region of the junctional portion of the tumour with epithelial gland-like spaces enclosed in an acellular mucoid matrix. Above is seen cartilage with its denser matrix. (x 80)

FIG. V. Photomicrograph of Case 2 showing a higher magnification of the epithelium and stroma seen in Fig. IV. (x 360)

Histologically the two nodules consisted almost entirely of hyaline cartilage enclosed by capsules of dense connective tissue. Between the capsule and the cartilage mass were several alveoli lined by a single layer of cuboidal epithelium and clumps of epithelial cells, both supported by a stroma of fibrous connective tissue (Fig. III). The cells were quite regular in arrangement and in the size and staining of both cytoplasm and nuclei. Progressive stages of transition from the adjacent tissue to the typical hyaline cartilage in the centre were observed. First, there was a change in the stroma which became more homogeneous, losing the sharp definition of its collagen bundles and tissue spaces, but leaving sharply demarcated lacunae which contained the epithelial elements. At this level, therefore, there were epithelial clumps and alveoli enclosed in a mucoid matrix. Nearer the middle the mucoid stroma was transformed into typical hyaline cartilage matrix (Figs. IV and V). Secondly, the cell groups began to break up into smaller aggregations, losing their previous alveolar arrangement so that finally each cell was separated from its neighbour by what was now true cartilage matrix. The cells were morphologically indistinguishable from chondrocytes seen in hyaline cartilage from the common sites. The surrounding breast tissue showed hyperplasia of both ducts and periductal connective tissue. The duct into which the larger nodule projected was lined by two layers of columnar epithelium and was a lactiferous sinus (Fig. VI).

Diagnosis. The diagnosis of fibroadenoma of the breast with cartilage formation was made.
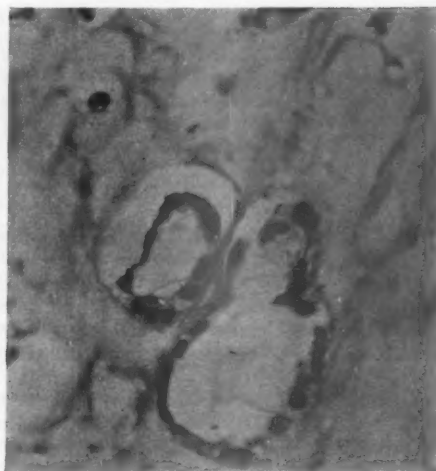

FIG. VI. Photomicrograph of portion of the collapsed cyst lining from Case 2. (x 80)

DISCUSSION

The formation of cartilage and bone in epithelial tumours is not uncommon. Their occurrence in "mixed" salivary tumours is well known but they are seen elsewhere, for example, in carcinomata of the stomach, gall-bladder and uterus (Willis, 1953) and in carcinomata of the rectum (Dukes, 1939).

The histogenesis of cartilage and bone in epithelial tumours has been the subject of much discussion. Allen (1940) recognized two distinct avenues of cartilage production in his malignant mammary tumours in dogs. In places there was gradual chondrification of the connective tissue stroma without participation of the epithelium, but elsewhere there was a gradual conversion of epithelium to cartilage by the following stages:

(i) loosening of the acinar epithelium;

(ii) isolation of the epithelial cells in a matrix so as to simulate myxomatous tissue;

(iii) collagenization of the mucoid matrix;

(iv) homogenization and lacunar formation.

This gradual conversion of epithelium to cartilage was seen to occur in the tumour from the second patient described above (Case 2). From the visual evidence provided by this case it was reasonable to deduce that here, as in Allen's cases, the cartilage was a direct product of the epithelium—with incorporation of isolated epithelial cells in the developing cartilage matrix until these cells were eventually indistinguishable from cartilage cells derived from the usual situations. The bone produced in his cases Allen ascribed to direct ossification of the stroma of the tumour but without epithelial participation. Clearly this was the method of formation of bone in Case 1 described in this paper where, prior to the appearance of calcium deposition and ossification, all epithelium had disappeared and the connective tissue stroma had become myxomatous. Calcification and ossification had then occurred in the mucoid stroma.

Not all writers have agreed with Allen's views concerning the formation of cartilage by epithelium. Tudhope (1939) described a mixed tumour of the breast containing areas of fibroadenoma, carcinoma, sarcoma and nodules resembling cartilage. There was a gradation from carcinomatous cells in clumps surrounded by a mucinous stroma to true cartilage with chondrocytes and also osteoid tissue. He claimed that the carcinoma cells had become embedded in the mucinous stroma and when that stroma had condensed a structural resemblance to cartilage was seen. This he called pseudocartilage. At the same time this change had induced the connective tissue stroma to produce true cartilage. Willis (1953) stated that the microscopical appearances of cartilage may be simulated by epithelial cells scattered in a mucinous secretion. However, bone or true cartilage often developed in the fibromatous tissue of the tumours by metaplasia.

No one has doubted the transformation of stromal connective tissues into cartilage in the vicinity of epithelium. The capacity of epithelium to induce metaplasia in connective tissues has been well recognized following the work of Huggins (1931). Huggins noted the development of bone in dogs in relation to epithelial cysts which arose when bladder mucosa was transplanted into the parietal tissues of the animals. A difference of opinion has arisen over the question of whether true cartilage or only a pseudo-cartilage can be formed by epithelium. The same problem has arisen with studies of mixed salivary tumours and cartilage-forming sweat-gland tumours, both of which may legitimately be compared with breast tumours as both breast and salivary gland are probably modified sudoriferous glands.

Simard (1938), in reporting a tumour with the structure of a "mixed salivary" tumour from the palm of the hand, described the changes we have seen in the breast with the transformation of imprisoned epithelial cells into true chondrocytes. On the other hand, Lennox et alii (1952) described 11 mucin-secreting hidradenomata of the skin, 4 of which possessed a typical mixed salivary tumour pattern and contained cartilage. Investigation of the mucins in these specimens showed that there was a progressive alteration in their properties, between the mucin in the epithelial cells, that in epithelial cysts and the mucin in contact with the stroma. He suggested a final change from stromal mucin into cartilagenous matrix, as this last was always surrounded by stromal mucin and often blended with it. The matrix, therefore,

was of epithelial origin, but although the context of the paper suggested that the cartilage cells were stromal in origin their origin was not definitely stated.

Willis (1953) referred to "cartilage-like" tissue in mixed tumours of salivary glands and stated that the properties of this tissue were not identical with true cartilage. This was in agreement with Fry (1927) who also wrote that there was no true formation of cartilage as the cells in the homogeneous dense matrix remained epithelial. The opposite view had already been expressed by Masson and Peyron (1914) who spoke of a complete transformation of epithelial cells to cartilage cells in mixed salivary tumours. Studies of experimental fowl carcinoma by Foulds (1937) led him to believe that two things occurred: the formation of true cartilage by connective tissue cells of the stroma and secondly the formation of cartilage-like tissue by imprisonment and modification of epithelial cells in a matrix of their own production.

There can be little doubt, however, that the tumour presented as Case 2 contained true cartilage. Furthermore there was visual evidence of the progressive change from epithelium to cartilage. The metaplastic production of cartilage is not, therefore, an accomplishment of connective tissues alone, but of epithelial tissues as well. The general notion of the development of cartilage from connective tissue has been gained from a study of embryology. Recent work in this field by de Beer (1947) has shown that the cartilage of the branchial arches of the axolotl is formed from the epithelium of the neural crest. This phenomenon is of fundamental importance when applied to pathology and in particular gives strong support to the proposition that cartilage can be formed by the epithelium of tumours.

Now let us consider the nomenclature which should be applied to this group of tumours. Had it been stated that these breast tumours had arisen in the testis they would have been called by some "teratomata," implying a simultaneous neoplastic change in, at least, three types of cell: chondrocyte, osteocyte and epithelial cell. If their origin had been parotid gland they would have been called "mixed" salivary tumours—the term "mixed" implying some special character of growth — but, because they were removed

from the breast where familiarity had not developed a special terminology, they were called fibroadenomata in which there had been formation of cartilage and bone. The acceptance of the concept of metaplasia has made it unnecessary to develop a new fundamental idea to explain the presence of many types of tissue in these tumours. As stated by King (1952), in reference to testicular "teratomata," it was probable that most were carcinomata showing multi-differentiation, while the benign examples were probably hamartomata. We happened to conform with this idea when the 2 cases which have been the basis of this study were called fibroadenomata of the breast containing metaplastic cartilage and bone. The terms "enchondroma" or "osteoma" of the breast would have been misnomers, as both would have drawn attention to an obvious but, from the point of view of classification, relatively unimportant character (indeed an accident) of growth. The tumours were both fibroadenomata.

In the present state of our knowledge it is not yet possible to be absolutely certain of the origin of cartilage in this group of tumours, but evidence is accumulating from both pathology and embryology, that cartilage may be formed by epithelial as well as stromal metaplasia.

SUMMARY

1. An indication of the rarity of cartilage and bone in breast tumours is given and the literature surveyed.

2. Two cases, one illustrating cartilage formation probably from epithelium and the other showing bone development in stroma, are described.

3. The histogenesis of cartilage and bone in these tumours and the related hidradenomata and mixed salivary tumours is discussed and the conclusion previously reached that cartilage may be formed by epithelial metaplasia as well as by alteration of the tumour stroma is confirmed. Bone is a product of stromal metaplasia.

4. The nomenclature of this group of tumours is discussed and the terms "enchondroma" and "osteoma" of the breast discarded in the classification of fibroadenomata of the breast containing metaplastic cartilage and/or bone.

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Books Reviewed.

CLINICAL GENETICS.

This volume, one of the smaller of the Brodingnagian newcomers to our library shelves, is a general review of the subject of genetics as applied to clinical medicine.

It is divided into two main parts. Section I is devoted to theoretical considerations and provides a good summarized statement of such problems as sex limitation, polygenic inheritance, evaluation of linkage and mutation amongst other questions. There are interesting chapters on twin studies and the biometric evaluation of findings. There is also a valuable discussion of experimental methods.

The second section is clinical and the various diseases, which seem to include most of those to which the human frame is heir, are considered for the most part under the various anatomical systems. The amount of information here is large, well collated and should be very valuable.

In general this book may be said to contain a great deal of information which will be valuable to the practitioner who is interested in the subject and wishes to refer to the recent additions to knowledge and opinions in this field. The sceptic will derive some degree of amusement from the naive assurance and certainty of many of the statements. One might perhaps wonder that some of the many writers of the individual chapters did not perhaps take some warning from the statement in the preface that "Nowhere in clinical studies, is theory—itself rapidly changing—so much in advance of empirical observations." Nevertheless in many parts of the book there is a scientifically critical attitude. In general this is a book to be recommended to those who are interested in this subject. A great deal of information is condensed in the small space (even though some of it does not obviously belong) and the viewpoints regarding recent ideas on genetics are well presented.

The general production of the book is good although the type of the paper, which results in an overall weight of nearly 3½ pounds, suggests a permanence which the volume does not obviously deserve. The illustrations are, for the most part, well produced and the tables are clear. The index is adequate.

PEPTIC ULCER.

In present day society peptic ulcer has assumed a place of great importance both as a cause of loss of working time and also for its fatal complications. No one can deny that there has been a real increase in incidence of the disease in the past fifty years—this has been amply proven. It is, however, humbling to realize that our only definite line of treatment is to employ a destructive operation that, except for a few modifications, was devised at the beginning of the century. From time to time new methods of treatment have occupied the centre of the stage (histidine, vagotomy and now anti-vagal drugs) but after a brief period of popularity, they have all suffered the same fate of eclipse. It is not quite fair to place the vagal paralysants in the same group as histidine, since they have been shown to affect the motility of the stomach and also to diminish the secretion of acid. Unfortunately the earlier claims for their ability to cause healing of the ulcer have failed to be substantiated in the majority of cases. This does not mean that progress is not being made in our understanding of the problem of ulcer. On the contrary a tremendous amount of time and energy has been devoted to this subject and the literature is full of reports on different aspects of the disease. Some of the most painstaking work has come from Professor Illingworth's department in Glasgow. It is therefore a pleasure to read a book written by the head of this department—written by a man with a vast experience of his subject.

This book is almost a summary of the modern ideas on peptic ulcer since it makes no attempt to the proportions of its American cousins. For this reason it should appeal to a wider reading public than do the more impressive tomes that have been published. Unfortunately in places, the chapter on peptic ulcer of the oesophagus for example, one is left with a feeling of incompleteness. The division into sections is a happy choice and for the most part these stand out very well as lucid interpretations of the many aspects of the disease and the factors leading to its production. The chapter on pain in peptic ulcer is a very good example of this—it gives a very balanced view on a subject that has been a battleground for conflicting views. Both sides of the question are fairly dealt with and criticisms are made of both theories but instead of leaving the matter completely undecided, the author advances a further theory to cover the best features of both schools of thought. This method of approach could well be taken as an example by other medical writers.

The changing of incidence of peptic ulcer is reviewed in detail with many excellent charts to illustrate this interesting aspect of the disease. As so much of this work has been done in Britain it is to be expected that it would assume its proper proportion in a British book. Interesting figures are also presented on the variation in perforation of ulcers and the great improvement in mortality rates from this complication. The physiology of gastric secretion and motility is adequately dealt with and a good list of references is given for those who require greater detail. It is a pity that the author confuses banthine with the hexamethonium group of drugs when dealing with gastric motility. It is also obvious

that he has not personally used the banthine group of drugs in his brief reference to their place in medical treatment. He has confused their side effects with those of hexamethonium. Further there is no description of their place in treatment nor what results can be expected. This is the penalty for one man, a surgeon, writing on all aspects of peptic ulcer. In fact the chapter on medical treatment is the weakest in the book, giving out-of-date emphasis to the sieving of vegetables and neglecting the proper role of the belladonna alkaloids. The danger of alkalosis with the use of absorbable alkalis is too summarily dismissed. The major criticism of the book is that the author does not make up his mind on some of the most important aspects of the disease and as a result vague statements creep in. This is seen in the indications for surgery in uncomplicated ulcer but particularly in bleeding ulcer. One is not quite clear if Illingworth still condones vagotomy at the end of this section although he appears to be against it at the beginning. In discussing hour glass deformity of the stomach indecision is again seen. It is unsatisfactory to say that hour glass deformity due to ulcer must be distinguished from that due to carcinoma without giving some detail.

In dealing with techniques the author has wisely kept to proven operations so that the work is not unduly burdened with technical procedures. It is a surprise to know "that anaemia after any type of gastrectomy can be prevented by thorough medication with iron." A statement that does not apply to a percentage of unfortunates who develop the malabsorption syndrome. This statement is repeated when dealing with the follow-up review of gastrectomies and causes a little hesitation in accepting the figures in their entirety. The chapter on stomal ulcer is very well done and gives a clear summing up of the problems involved. Not all surgeons would agree with the support for gastro-enterostomy in females.

Despite some criticism this volume is an excellent review of the subject, presenting some material in collected form for the first time. The illustrations with a few exceptions are good and the painting of gastroscopic appearances are excellent. It is not a reference book but one that should be read by anyone who has an interest in the problem of peptic ulcer and that should be most physicians and surgeons.

STUDIES IN FLUID BALANCE.

By PETER S. HETZEL, M.D., B.S.(Adel.). Adelaide, Australia: K. M. Stevenson, Government Printer, 1952. 8½" x 5½", 183 pp., 26 figures, 21 tables.

The volume of 183 well printed and moderately well illustrated pages is essentially a record of the author's studies in the field of fluid and electrolyte balance.

It is divided into five main sections:—

(I) "A Review of Present Knowledge of Electrolyte and Water Metabolism." This section (22 pages) is a short review of most of the main essentials of this very complicated subject up to the time of publication. The review has been more from a theoretical viewpoint than from the everyday practical clinical one, so that the reading may appear complicated and frightening to one not used to handling these problems.

(II) "Studies in the Metabolism of Water and Extra-Cellular Ions." Here various "pure" syndromes are discussed and illustrated by the author's own cases. To those conversant with the subject it is an interesting section, but it is disappointing to see "pure" syndromes illustrated by what are obviously "mixed" fluid and electrolyte syndromes. Such illustrations may mislead the non-critical reader. Complete electrolyte patterns are not quoted, this allows the reader no check on the accuracy of the figures. Since serum proteins are quoted in gms.% instead of in m.eq./litre, one wonders what check the author had. The absence of bicarbonate estimations is often notable since this ion gives the best clinical clue as to blood pH. The handling of an intestinal obstruction with a low serum sodium and a low serum bicarbonate is quite different from that when the bicarbonate value is normal or raised. Apart from the above, the propositions put up are academically, fairly sound.

(III) "Studies in the Metabolism of Potassium as the Chief Intra-cellular Ion." On reading this section one tends to be left with the impression that the practical application of potassium therapy is very complicated. This is a misleading view.

(IV) "Studies on the Excretion of Electrolytes after Operation." Since publication of the book more complete works by other authors are available. They bring the patterns into a more concrete perspective than the rather tentative views put forward here.

(V) "The Application of Intravenous Therapy in Surgery." Many of the views regarding the individual intravenous fluids are not generally accepted either theoretically or practically, by others in this field. The undue emphasis on "normal saline" is surely a relic of the past—and throughout the ambiguous use of the term "saline" is to be deprecated.

The section on "Certain Forms of Shock" is untidy and jumbled, while that on "post-operative shock" indicates to me the author's lack of experience in its treatment.

The whole book is of some interest to those already conversant with the subject, but it does not provide a simple and easy guide for the average practitioner.

EAR, NOSE AND THROAT DISEASES.

By WILLIAM McKENZIE. Edinburgh: E. and S. Livingstone Ltd., 1953. 8½" x 5½", vii plus 260 pp., 95 illustrations. Price: 21s. net

It is refreshing to read a textbook written for students by an author still retaining, and communicating, a youthful freshness and enthusiasm. The book more than adequately covers the necessary field: the illustrations are excellent and much trouble has been taken to make them clear and explicit. The section on tonsils and adenoids is excellent and rigidly stresses the place of a careful history in the decision as to whether to operate or not. The treatment of secondary haemorrhage is, however, far too sketchy.

The book can be wholeheartedly recommended for the use of students, and those in general practice will indeed be well equipped if they utilize all that is to be found between its covers.

THE ECZEMAS—A SYMPOSIUM BY TEN AUTHORS.

By L. J. A. LOEWENTHAL, M.D., M.R.C.P., D.T.M. and H. Edinburgh: E. and S. Livingstone Ltd., 1954. 9½" x 6", vii plus 267 pp., 77 illustrations. Price, 35s. net.

The monograph is a deservedly popular form in medical literature at the present time for presenting a subject in which fundamental changes of outlook are occurring as new knowledge is gained in the clinic and laboratory. In it one can expect an integrated, comprehensive and authoritative survey of more than transient value.

Such a review of "The Eczemas" is undoubtedly needed at present, and the present volume succeeds in meeting it in spite of certain difficulties. It is the result of an international collaboration by ten dermatologists in Europe, America, Britain and South Africa. Thus it is not surprising to find some degree of fragmentation of the subject matter and some differences in approach among the contributors although it ensures their high calibre. Furthermore, eczema and dermatitis is a diverse family, so that drastic compression has been necessary to reduce all aspects to 236 pages of text and yet avoid the charge of superficiality. However the result is highly creditable, in producing a stimulating and useful book, and the general unity of the subject has not been impaired.

There are 13 chapters, covering all the major types of eczema, and also diagnosis and treatment. The chapter on allergic eczema ably presents the experimental and clinical observations which have so greatly influenced the modern outlook on pathogenesis. The remainder of the book emphasizes practical aspects without being empirical. The chapters on atopic dermatitis, contact eczema, histopathology, the role of bacteria, and eczematous eruptions with special features are clear and well balanced. Psychiatric aspects have not been allotted a chapter, on the ground of lack of precise information, and few will disagree with the omission.

The editor's chapters on the phenomenon of dissemination, diagnosis and principles of treatment are sound and practical.

Lists of substances and recommended concentrations for patch and scratch tests, and a formulary of 35 prescriptions, add to the practical value of the book.

The 77 illustrations are well chosen and beautifully reproduced, and the general production is of the high standard consistently maintained by Messrs. E. and S. Livingstone Ltd.

STONE IN THE URINARY TRACT.

By H. P. WINSBURY-WHITE. Second Edition. London: Butterworth and Co. (Publishers) Ltd., 1954. 9½" x 6½", ix plus 342 pp., 144 illustrations.

This book is a re-arranged and re-written presentation of the author's earlier work. The first edition was published in 1929, although there is no indication whatever in the present volume of this date.

The book contains a large amount of information, as would be expected from a surgeon of St. Peter's and St. Paul's Hospitals. As he states, it is built on experience of 866 personal cases of urinary calculus.

The whole subject of calculi in the urinary tract has been dealt with and interesting information will be found about the less usual varieties such as prostatic and preputial stone. The book is clearly the work of a practising surgeon and it is in the special sections that information of value is to be obtained.

As far as the fundamentals of the subject are concerned the book definitely lacks cohesion and insight and for this reason cannot be recommended to students. In devoting 30 pages to the aetiology of urinary calculi the author states: "After having considered so many factors which are involved in the aetiology of renal calculus, the subject is apt to appear rather confused." It does!

The analyses of actual observations and operative findings and the like are of definite value and will be found of use to the urologist. In general the illustrations are well chosen and though drawings are more generally used than photographs they illustrate the features indicated in the text. The text is produced in clear type on heavy art paper and there is an adequate index.

METHODS OF EXAMINATION IN EAR, NOSE AND THROAT.

By W. G. SCOTT-BROWN. London: Butterworth and Co. Ltd., 1954. 8½" x 5½", ix plus 110 pp., 94 illustrations. Price: 28s.

"Methods of Examination in Ear, Nose and Throat," by W. G. Scott-Brown, is an excellent book of one hundred pages, which must attract not only the student and general practitioner but, to a considerable extent, the specialist, particularly the demonstrator in this subject.

This book is concise and very clearly written. It is fully illustrated with photos, sketches, and reproduction of X-ray photos. It contains the many small details in method of examination which enable the practitioner to obtain a detailed view of areas sometimes so difficult to demonstrate.

The book is divided into six chapters, the first four embracing the full physical ear, nose and throat examination; the last two embracing the functional examination of the cochlear and vestibular apparatus.

In general equipment one applauds the author's insistence on suction apparatus for ear and nose examination—an extremely useful instrument which one hopes may become more commonly used in this country.

Examination of nose and nasopharynx is detailed and thorough but many must read, for the first time, of the digital examination of the post-nasal space through the soft palate, although one agrees heartily that the space should never be examined digitally without general anaesthesia. One also agrees that the pharyngoscope is almost an essential instrument.

The description of examination of sinuses is good, and covers the full scope of the specialist. The detail of the procedures is well written and illustrated but perhaps there might be some warning about the routine antral puncture and the much more difficult sphenoid puncture. Canulization of the normal ostium is not mentioned.

Examination of the larynx is well described, but one feels that the use of a local anaesthetic spray, such as decicaine, could be mentioned and recommended, to enable an easier and more precise examination to be made.

Direct examination of the larynx, with laryngoscope, is well given—especially one agrees with the general anaesthetic technique. The sketches 54, 55, 56 appear to me to differ from the accepted technique of handling the laryngoscope, as described by Jackson.

The ear is very well covered, but one feels there could be a warning in such a book as this about the difficulty of removing wax which may be impacted, and the dangers of use of the blunt hook, as illustrated. The description of the appearances of the drum one feels could be considerably augmented.

The functional examinations of the cochlear and vestibular apparatus are extremely well covered. The ability to cover this large field in a book of this size is almost a masterpiece of description, and fully covers the everyday knowledge of the specialist. These chapters give an excellent summary of the latest work done in interpretation of the caloric and hearing tests.

One can only unreservedly recommend this book to all those who are in any way interested in otolaryngology, and one can congratulate the author on a book which should be on the shelves of all those who practise otolaryngology in part, or as a speciality.

new both in principle and design, already becoming widely accepted and used, while the context of the book deals solely with the principles and use of the old. Lest he be tempted to regard the work as already outmoded, the reader should accept the advice of the editor in his foreword and regard the book as "the interim report of a bold long-term clinical trial." Its real merit and significance is then revealed.

The section dealing with the management of osteo-arthritis of the hip is excellent — in fact it is probably safe to say — unsurpassed in modern orthopaedic literature. Proper emphasis is laid upon the importance of careful selection of patients for operation, pain of course being the major—in fact almost the only—indication. A rather novel method of assessment before and after operation is admitted by the authors to be perhaps "unduly complicated and even tiresome." While it has the advantage of a common basis for comparison, not only before and after treatment but also for different clinics, it tends to ignore the all important human factor when adding up the "score." Descriptions of operative technique are clear and concise. It is, however, doubtful if the photographs of actual stages in the operation, so faithfully reproduced on pages 24-41 inclusive, would be intelligible at all were it not for the beautifully drawn black and white key sketches which accompany them. The photographs would not be missed if omitted in future editions.

Fractures of the neck of the femur are faithfully dealt with and few who essay this type of surgery can fail to learn much from what is written. Subsequent chapters are at most, of more academic than practical interest, since the series presented by the authors are admittedly too small to warrant conclusions other than that results to date show considerable promise. Few orthopaedic surgeons however will share the author's faith in a few cancellous chip grafts when the stem of the prosthesis is too loose or the reamer perforates the floor of the acetabulum.

Lest it be thought that these strictures are too harsh, let it be clearly stated that this monograph represents the very latest and best in orthopaedic practise in relation to the very complex problems presented by the painful hip joint. The admiration of the whole surgical world belongs to the Judet brothers and their confreres, not least of these being the Messrs. Drapier, whose superb instruments, illustrated in this volume contribute so much towards simplication of this most difficult branch of surgery.

RESECTION-RECONSTRUCTION OF THE HIP.
Arthroplasty with an Acrylic Prosthesis.
    Edited by K. I. NISSEN. London: E. and S. Livingstone Limited, 1954. 8½" x 7", xii plus 151 pp., 101 illustrations. 30/- net.

The first impression after reading this admirably produced monograph is that its authors have become slightly too enthusiastic about their theme. It is perhaps unfortunate that, in a preface to the new English edition edited by K. I. Nissen, considerable prominence is given to an improved prothesis, partly

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